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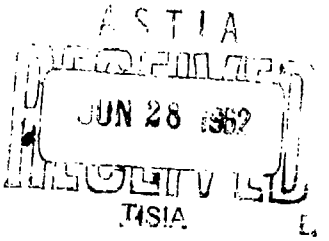

DEFENSE ATOMIC SUPPORT AGENCY

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BIOLOGICAL EFFECTS OF BLAST

by

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Presented before
The Armed Forces Medical Symposium
Field Command, Defense Atomic Support
Agency, Sandia Base, Albuquerque, New Mexico
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FOREWORD

This report deals with the physical and biological aspects of blast phenomena and represents a selective summary of the current status of knowledge regarding the Biological Effects of Blast. The material was presented before the Armed Forces Medical Symposium held November 27 - December 1, 1961 at Sandia Base, Albuquerque, New Mexico, under the sponsorship of the Field Command of the Defense Atomic Support Agency of the Department of Defense.

The data are useful to civilian and military personnel interested in that aspect of environmental medicine concerned with the biological consequences of exposure to blast phenomena, particularly with reference to nuclear weapons and explosives. However, much of the information also refers to conventional high explosives and therefore construction companies, manufacturers of explosives and organizations handling nuclear weapons will find the material of value. The biological criteria noted, along with the estimates of human tolerance, must be regarded as tentative and subject to the limitations which bear upon all attempts to assess human response from the extrapolation of interspecies data obtained during animal experimentation. Finally the material is not all inclusive and those requiring the broadest possible knowledge are referred to the bibliography for detailed data.

ABSTRACT

The current state of knowledge relevant to biological blast effects was summarized in a selective manner. Initially, five problems of concern to those who would relate the environmental variations produced by nuclear weapons with biological response and hazard assessment were pointed out. Primary, secondary, tertiary, and miscellaneous blast effects were defined and selected interspecies experimental data of a physical and pathophysiological nature useful in estimating human response were presented. Tentative biological criteria defining "safe" levels of exposure were set forth as were survival curves for different conditions of exposure in Hiroshima. These were discussed along with the comparative variations in range of the "free-field" effects as they vary with explosive yield. The fundamental requirement for surviving seconds, minutes, and hours to abet survival for days, weeks, months, and years was emphasized along with the necessity for planning protective measures against all hazardous weapons effects as one attractive alternative for minimizing casualties and maximizing survival in the event of a nuclear war.

ACKNOWLEDGEMENTS

The data reported have been the result of a consistent effort in contract research since about 1951, made possible by continuous support from the Civil Effects Test Organization and the Medical Branch of the Atomic Energy Commission's Division of Biology and Medicine, along with a few funds from the Federal Civil Defense Administration and the Armed Forces Special Weapons Project up to about 1959. Following this date, for a year the Defense Atomic Support Agency joined the AEC in sponsoring the work, and since October 1960, the DASA has funded the effort in Blast and Shock Biology while the CETO of the Division of Biology and Medicine has provided support for activities in the broader area of Weapons Effects.

The research effort involved a team of Lovelace Foundation personnel, and it needs be clear that the writer acts as their representative. The work has been carried out mostly by individuals working under the leadership of Dr. Donald R. Richmond, Head of the Foundation Department of Comparative Environmental Biology; Mr. I. G. Bowen, Head of the Department of Physics; Mr. R. V. Taborelli, Head of the Department of Engineering; and Dr. Thomas L. Chiffelle, Head of the Department of Pathology.

Appreciation for editorial aid is tendered to Mrs. Ruth P. Lloyd, who along with Mrs. Vicki Newsom, Mrs. Helen T. Vatoseow, and Mrs. Isabell D. Benton, typed the rough and final manuscript. Also, thanks are due Mr. Robert W. Smith, Mr. George A. Bevil and Mr. Edward M. Johnson, who processed all the illustrative material.

Finally, the writer wishes to acknowledge the interest and encouragement of Major General Harold C. Donnelly, USAF, Commander, Field Command, Defense Atomic Support Agency, Sandia Base, Albuquerque, New Mexico, along with the help and cooperation of his staff, particularly, Colonel Savino W. Cavender, (MC), USA, Surgeon, Field Command, under whose immediate direction the Armed Forces Medical Symposium was organized and arranged.

INTRODUCTION

Before turning specifically to the assigned subject of "The Biological Effects of Blast", I wish to say several things.

First, a common sense approach to assessing biological weapons effects dictates that one must recognize all environmental variations which alone or in combination pose a hazard to man. A balanced concept in this regard must view both direct and indirect effects as well as the immediate, intermediate, and long term consequences of nuclear explosions.

Second, it also makes common sense -- in fact, it is essential and necessary -- to look upon survival as a stepwise process that first requires survival for time periods like milliseconds, seconds, and minutes before survival for hours, days, and weeks can be realistically contemplated. Likewise, it makes little sense on a national scale to work upon survival over months and years without first assuring that lethality in the earlier time period is minimized; i. e., in a manner of speaking, one must "earn" the opportunity to survive over the long term by doing those things that maximize survival over the short and intermediate time periods.

Third, while such thinking in case of a nuclear war applies to all inhabited areas as far as biological hazards are concerned, they apply particularly to cities and urban complexes where population densities are high, for it is here that the immediate effects will take their greatest human toll.

Fourth, the state of preparation of a population can make great differences in survival -- as will be emphasized later -- and few will challenge the desirability of maximizing the chances for survival of

people and the nation. To many, this means fallout shelters for protection against residual radiation. To a growing number — as yet all too few — this also means protection against the more immediate effects as well, and I am among those who believe that blast, thermal, and initial radiation will depress over-all survival much more than will the consequences of exposure to residual radiation. I agree that all should have fallout protection. I contend that this is not enough for the free world and a nation as wealthy as the United States, or for any nation seriously depending for its security on nuclear arms. The nature of the immediate hazards in highly-populated areas demands recognition and there needs be a rational balance in planning and implementing measures to enhance survival on a national scale.

Fifth, it is well now to state candidly that anyone undertaking a discussion of even limited aspects of environmental medicine as it relates to nuclear explosions faces a difficult task indeed. Whatever is said over a two-hour period must of necessity be highly selective. Just to talk about the physical and biological aspects of blast involves a host of bio-physical and physical parameters ranging from weapons phenomenology and the modifications of "free-field" effects by the conditions of exposure, to the etiologic events which begin with the transfer of energy to a biologic target, and end when the biologic response is complete.

Sixth and last, by way of introduction, let me say what will be discussed over the next 30 minutes:

1. First, five problem areas that more or less generally concern those dealing with any of the several biological effects will be noted;

2. Second, the scope of blast biology will be defined;

3. Third, mention will be made of recent work of a physical nature which contributes to understanding the environmental variations produced by explosive events;

4. Fourth, the significant biological consequences of exposure to blast phenomena will be discussed;

5. Fifth, selected information from the Japanese experience in 1945 will be presented; and

6. Sixth and last, if time permits, a few remarks will be made about scaling the major "free-field" effects as they vary with yield and range, the significance such data have in assessing over-all hazards, and the mandatory need that exists for making the practice of blast, radiation and thermal prophylaxis a reality.

I. Problem Areas Relevant to Biological Effects of Nuclear Explosions

Those interested in any of the major weapons effects face formidable problems of great significance in at least five areas. These will now be presented.

Table 1 directs attention first to the necessity of understanding the source of the environmental variations produced by nuclear explosions. There are variations in effects which, among other things, depend upon weapon design, yield, burst conditions, range, and weather. Within certain limits, information is available in such books as The Effects of Atomic Weapons, The Effects of Nuclear Weapons, and various manuals which allows one to set forth the magnitude of each effect – the "dose", if you will – as this varies with range and explosive yield, assuming the absence of buildings and a flat terrain. This exercise is often termed "free-field" scaling.

However, attenuation or augmentation of the major effects may well occur depending upon how the conditions of exposure influence or modify the "free-field" values. This, I call "geometric scaling". Biomedically, it is hardly enough to know the hazardous environmental variations in the general region of an individual's house, office, or place of work. Rather, it is necessary to know the magnitude of the environmental variation at the location of the biological target. This point is important and deserves great emphasis. As will be seen later, there may be a great difference between what occurs outside a building on the one hand and what transpires inside the structure on the other.

TABLE I

Problem Areas Relevant to Biologic Effects of Nuclear Weapons

Source	Design Yield Burst conditions Range Weather	"Free-field" scaling of major effects.
Attenuation and Augmentation	Modification of "free-field" phenomena by geometric conditions of exposure	"Geometric" scaling
Physical Interaction	Energy transfer to: physical objects and biological material	Secondary events
Biophysical Interaction	Energy dissipation by or within biologic targets	Etiologic mechanisms
Biologic Response	Major syndromes for isolated individual effects and combined injury	Hazard assessment

Table 1 points out three other problem areas. First, interaction can occur between the "free-field" effects and the materials which define the environment of exposure, and energy is often transferred to physical objects and biological media. These secondary events encompass fires, the movement of debris (such as glass missiles, bricks, sticks, and stones), and also the physical displacement of biological targets, which may be hurled bodily through the air.

Second, biophysical interaction transpires a process whereby energy is dissipated by or within biological targets. Such events are often fundamental in spelling out the etiologic or casual mechanisms at play within the living organism which are responsible for pathology.

Third and last, there is the problem area of biologic response. This encompasses the major syndromes associated not only with each isolated effect – the signs and symptoms of exposure to ionizing radiation, to blast, and to thermal energy, for example – but also what transpires when exposure to more than one or all effects occurs. The latter includes the multiple injury problem, about which all too little is known biologically. However, it is in this area of biologic response incorporating quantitative information about 'dose" and effect that one must work to assess hazards precisely. Here one desires to know what level of a specific environmental variation is relatively "safe", what level produces casualties, where does mortality begin, and at what level lethality is likely to be noted 100 per cent of the time.

All reasonably perceptive students of biologic weapons effects recognize the need for paying attention to the five problem areas just mentioned; they also recognize not only the many complexities involved, but the highly specialized knowledge that is required to discuss even one of the areas reasonably well. Too, there is the simple fact that much-needed empirical information simply is not yet at hand.

Since these things are so and one's knowledge is beset with many uncertainties, it is with a great deal of humility that I attempt to go further

with the discussion of even the relatively limited area of blast biology. However this may be, there is indeed a great deal of information available, and now the task is to keep the problem areas just considered in mind, while the discussion proceeds in a selective manner.

II. Scope of Blast Biology

The over-all scope of what has come to be called blast biology[1-4] can be stated briefly and somewhat arbitrarily as follows:

A. Primary blast effects are those associated with variations in environmental pressure which follow explosive events.

B. Secondary effects are those which transpire from the impact of debris energized by blast pressures, winds, ground shock, or gravity. Such debris or missiles may or may not perforate or penetrate a biologic target.

C. Tertiary effects encompass the consequences of gross bodily displacement of biologic media by blast winds, ground shock, and gravity.

D. Miscellaneous effects include:

1. The effects of exposure to dust, radioactive or not;

2. Non-line-of-site thermal burns apparently due to hot gases and dust; but may also involve the impact of hot objects, and

3. Blast-induced fires in contrast with those caused by the initial thermal pulse.

III. Physical and Biophysical Factors

It is apparent from the defined scope of blast biology that one must consider whatever data of a physical nature are needed to quantitate the environmental variations that occur in the immediate vicinity of the target, including the displacement of objects, be these missiles or man, if a relation with biological response — due mainly to dynamic accelerative or decelerative loading — is to be established. A few of the more important relevant matters will now be noted.

A. The Pressure Pulse - Most individuals are more or less familiar

with the wave of overpressure that eminates radially from an explosive source accompanied by blast winds of considerable force[5,6]. The magnitude and duration of the overpressure are functions of the yield and range, and both decay, though the pulse grows in duration as the wave moves away from the exploding fireball. The shape of the wave form as it decays with time and range is, under certain circumstances, markedly a function of burst height, terrain, and the temperature of the latter. However, for high air bursts and at some range from low air or surface bursts, the wave form is "clean" or "classical", meaning that a pressure gage, side-on to the advancing pulse will record pressures that rise sharply — almost instantaneously — to a maximum, and then show a decrease with time to reach a minimum which is below the previous ambient. After this, the underpressure will rise to reach the previous level. "Unclean" or non-classical wave forms[7] sometimes occur "free-field" and the rate of pressure rise is degraded, an important fact biologically, as will be stressed later.

B. Pressure Reflections - Should a "sharp"-rising pressure wave — the incident pulse measured in psi side-on to the advancing wave—strike a solid object like a wall placed across its path of travel, pressure reflection will occur[5,6]. This will be maximal if the angle between the pressure pulse and the object is 90°. The overpressure may increase to double or much more depending mostly on the magnitude of the initial pulse[8,9] and whether or not a shock wave comprises the leading edge of the pressure pulse; i. e., whether or not the wave form is "classical".

Also, a "free-field" pressure pulse may spill through relatively small openings into a large building or subway, and the resulting maximal pressure inside may be much less than that which momentarily existed outside[1,7]. On the other hand, depending upon the circumstances involved, the inside pressure may be magnified, may involve multiple reflections, or may not be significantly changed. These data place emphasis upon the fact that the pressure environment in the vicinity of a biological target and, therefore the biological response, is markedly sensitive to the geometry

of exposure; i. e., to the differences made clear by separating "free-field" and "geometric" scaling.

C. Blast Winds – Dynamic Pressure (Q) - Mention has been made of the blast winds which accompany the pressure pulse. These exert a force termed the dynamic pressure or Q on any object, the magnitude of which is equal to the difference between the face-on and side-on pressure[5, 6]. At a local static pressure of 5 psi from a nuclear weapon, this force is equal to near 0.7 psi and equivalent to maximum winds in excess of 160 miles per hour[6, 10] when the wave form is "classical".

Under some circumstances when atypical wave forms occur "free-field", the dynamic pressures can be much higher. For example, at a station where 6.6 psi was measured in Nevada, a dynamic pressure of 15.8 psi was recorded on one occasion[10]. The equivalent wind velocity was over 650 miles per hour. Also, high winds occur when blast pressures spill through openings in buildings where windows and doors fail. The wind direction may be positive or negative and endure for times near those of the over and underpressure[1, 7]. However, when pressures enter through openings into closed spaces, high winds exist at such openings and these endure only for the "fill-phase" of the structure; i. e., until the inside and the outside pressures become equal. As will be noted later, these winds can be extremely hazardous.

D. The Pressure-duration Relationship - The relationship between the magnitude and the duration of the overpressure is important. As was noted earlier, for each yield, the overpressure falls with range and the duration increases. For low yields, such as occur with conventional explosives, 100 psi may be associated with overpressure durations of about 2 and 10 milliseconds for charges as small as 50 and 4000 pounds, respectively[9]. In contrast, for yields of 1 kiloton (= 1000 tons of high explosive), pulse durations in the order of 100 milliseconds occur, while those for 1 and 10 megatons are about 1 and 2.2 seconds, respectively[6]. The point is, for the yields practical with high explosives in a military operation, the pulse duration for a given overpressure is very, very short;

whereas for nuclear explosives, the durations are very, very long for the same overpressure. There are at least two reasons for the significance of the pulse duration which will now be noted.

The first concerns the relation to the physical length of the overpressure pulse and the size of the biological target. For example, a pulse of overpressure traveling at near the speed of sound for standard conditions, say about 1000 feet per second, will pass a monitoring gage on the ground in 1 millisecond. If this is the case, the pressure pulse is 1 foot long. Another pressure may pass the gage in 100 milliseconds and will cover about 100 feet of the ground surface. A biological target the size of a cow, in the latter case, will be engulfed in the pressure field and "squeezed" for a considerable time. A 1-millisecond, 1-foot-long pressure pulse, on the other hand, can pass over the target and, because of the great difference in dimensions, only a small portion of the target at any one time will be covered and "squeezed" by high pressure gases.

The second reason concerns the displacement of objects by blast winds. Short-duration overpressures are accompanied by winds of short duration, and the period the blast winds have to accelerate an object is much shorter than is the case for long duration pressures and winds. In the latter case, much higher displacement velocities are likely to be attained.

The physical problem of displacement of objects will now be discussed in more detail. Concerned are special aerodynamic events whereby energy is transferred to movable objects.

E. Displacement - Fortunately, the physical parameters responsible for energizing objects as small as tiny pieces of glass and as large as man are generally similar, and determining the velocity-time and velocity-distance relationships for missiles and man can be discussed together as the following highly-simplified explanation will illustrate.

Consider Figure 1 which was computed for 5 psi overpressures produced by about a 30-kiloton detonation at Nevada altitude[11]. There are several interesting matters portrayed by the figure which will now be pointed out.

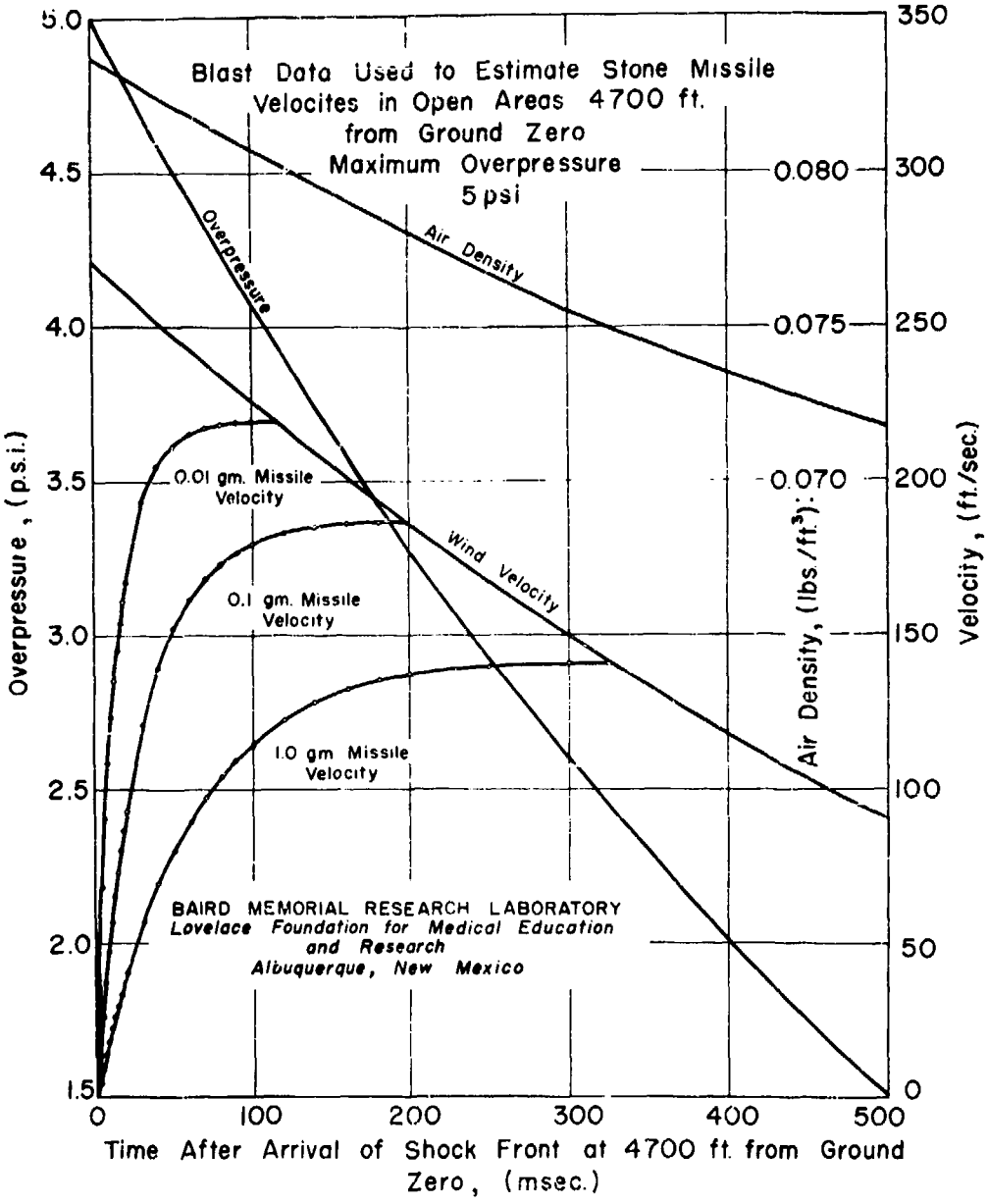

Blast Data Used to Estimate Stone Missile Velocites in Open Areas 4700 ft. from Ground Zero
Maximum Overpressure 5 psi
Overpressure
Air Density
Wind Velocity
0.01 gm. Missile Velocity
0.1 gm. Missile Velocity
1.0 gm Missile Velocity
BAIRD MEMORIAL RESEARCH LABORATORY
Lovelace Foundation for Medical Education and Research
Albuquerque, New Mexico
Overpressure, (p.s.i.)
Air Density, (lbs./ft.³)
Velocity, (ft./sec.)
Time After Arrival of Shock Front at 4700 ft. from Ground Zero, (msec.)
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
0.080
0.075
0.070
350
300
250
200
150
100
50
0
0
100
200
300
400
500

Fig. 1

Note the overpressure curve showing the decay of pressure with time using the overpressure scale on the left and the time scale on the bottom; also, note the wind-velocity curve as it decays with time using the right-hand scale. The three curves at the lower left of the figure portray the gain in velocity of stone missiles as a function of time after the arrival of the blast winds. The lightest missile of 0.01 grams, as shown by the top curve, gains velocity faster than the heavier 10-gram missile depicted by the lower curve. The maximum velocity in each case occurs at that instant the missile velocity becomes equal to the corresponding wind velocity.

Larger, heavier objects, such as man, take much longer to reach the velocity of the wind[2, 12]; maximum velocity in these cases is quite sensitive to pressure and wind duration and, therefore, to explosive yield. This is far less the case with small, light objects.

Now the time scale at the bottom of the figure properly can be replaced with a distance-of-travel scale, and it becomes clear that small, light objects travel shorter distances to reach maximum velocity than do larger, heavier objects.

This velocity-distance-time relationship can be further illustrated by noting data obtained photographically for a 165-pound anthropometric dummy exposed back-on to 5 psi produced by about a 40-kiloton detonation in Nevada.

Figure 2 shows the measured velocity-time curve[10, 12]. Note that a maximum velocity of 22 feet per second was reached in about 0.5 seconds at which time the dummy had moved a little over 8 feet. In 0.1 second, the dummy was moving 13 feet per second and had traveled about 0.9 feet. It is thus clear that when considering the velocity of objects energized by blast winds, one must also specify the distance traveled at least up to maximum velocity.

Experience with over 20,000 missiles at Nevada which were trapped, recovered, weighed, and their impact velocities determined at various

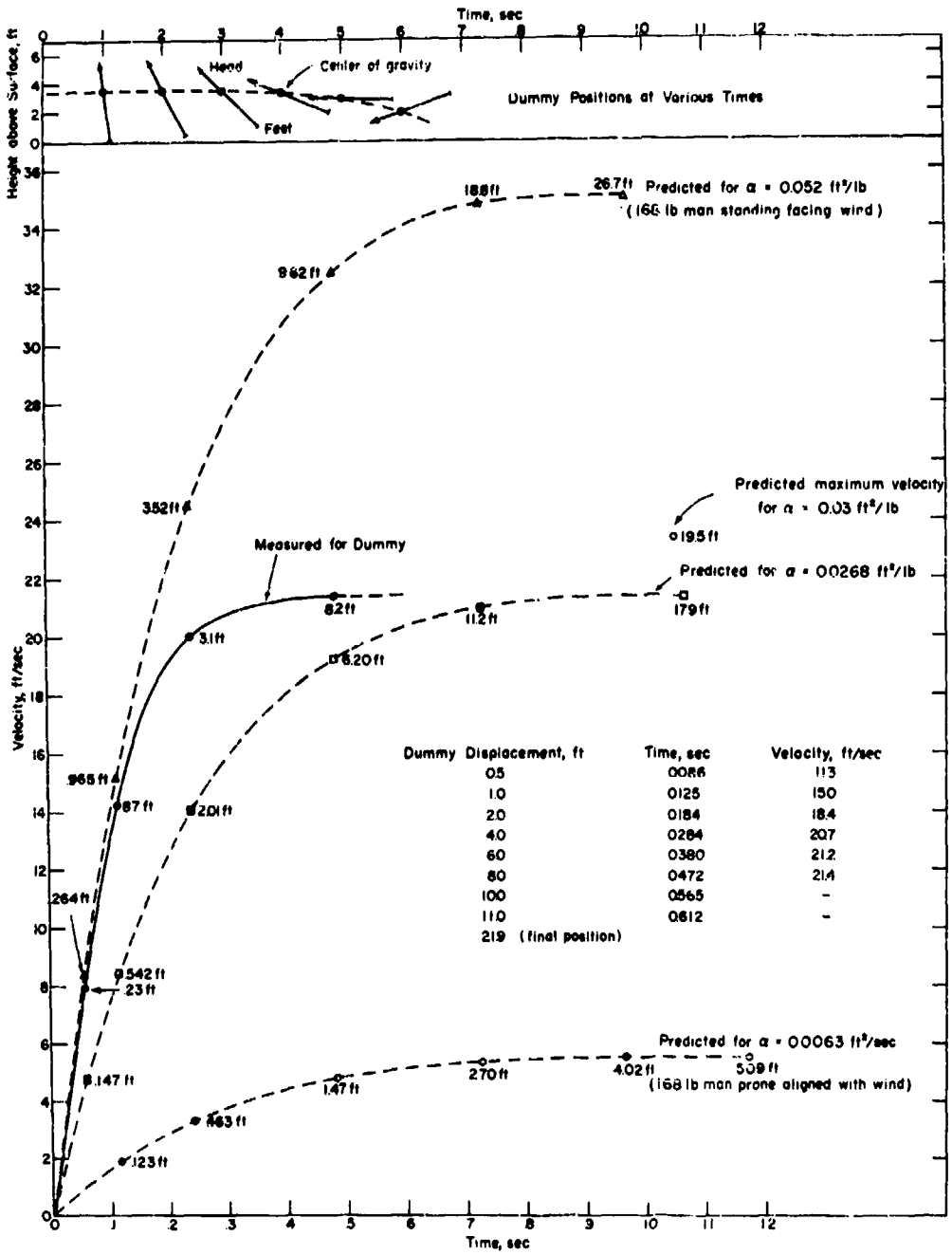

Time, sec
Height above Surface, ft
Head
Center of gravity
Feet
Dummy Positions at Various Times
Predicted for α = 0.052 ft²/lb
(166 lb man standing facing wind)
Predicted maximum velocity
for α = 0.03 ft²/lb
Predicted for α = 0.0268 ft²/lb
Measured for Dummy
Predicted for α = 0.0063 ft²/sec
(168 lb man prone aligned with wind)
Velocity, ft/sec
Time, sec
Dummy Displacement, ft | Time, sec | Velocity, ft/sec
0.5 | 0.086 | 11.3
1.0 | 0.125 | 15.0
2.0 | 0.184 | 18.4
4.0 | 0.284 | 20.7
6.0 | 0.380 | 21.2
8.0 | 0.472 | 21.4
10.0 | 0.565 | -
11.0 | 0.612 | -
21.9 (final position)

Fig. 2

distances of travel, has allowed Bowen to formulate a mathematical model[12] which allows velocity-time and velocity-distance predictions to be made for clean wave forms as these are influenced by range and explosive yield.

One parameter appearing in the relevant equations of motion is the acceleration coefficient, alpha, which is defined as a value in square feet per pound, equal to the product of the area of a displaced object and its drag coefficient divided by the mass.

$$(\alpha = \frac{A \cdot C_d}{m})$$

This will be discussed somewhat more by noting the top of Figure 2 which shows the position assumed by the dummy referred to previously. If one uses the acceleration coefficient of about 0.052 referable to the initial position of the dummy, the top dotted curve is obtained. If one uses an alpha of near 0.03, the second dotted curve is predicted. This works well for predicting maximum velocity but underestimates the velocity obtained in the early time periods for which the alpha referable to the initial position is preferable.

If, however, one corrects the alpha periodically for the changing positions of the dummy, the results shown in Figure 3 can be obtained[13]. The correspondence between the measured and computed velocity-time curve is quite good and lends considerable confidence to the prediction procedure.

The figure also presents the acceleration-time curve which shows a maximum "instantaneous" acceleration of about 4.5 G units. This is a tolerable load and points out that if a hazard exists under such circumstances it is not associated with the process of "getting going", but rather with the process of stopping. That this indeed can be dangerous will be alluded to later.

Now consider Figure 4 which was prepared by Bowen[13] following the completion of the laboratory studies undertaken to experimentally determine acceleration coefficients of different objects[14]. Alpha values are

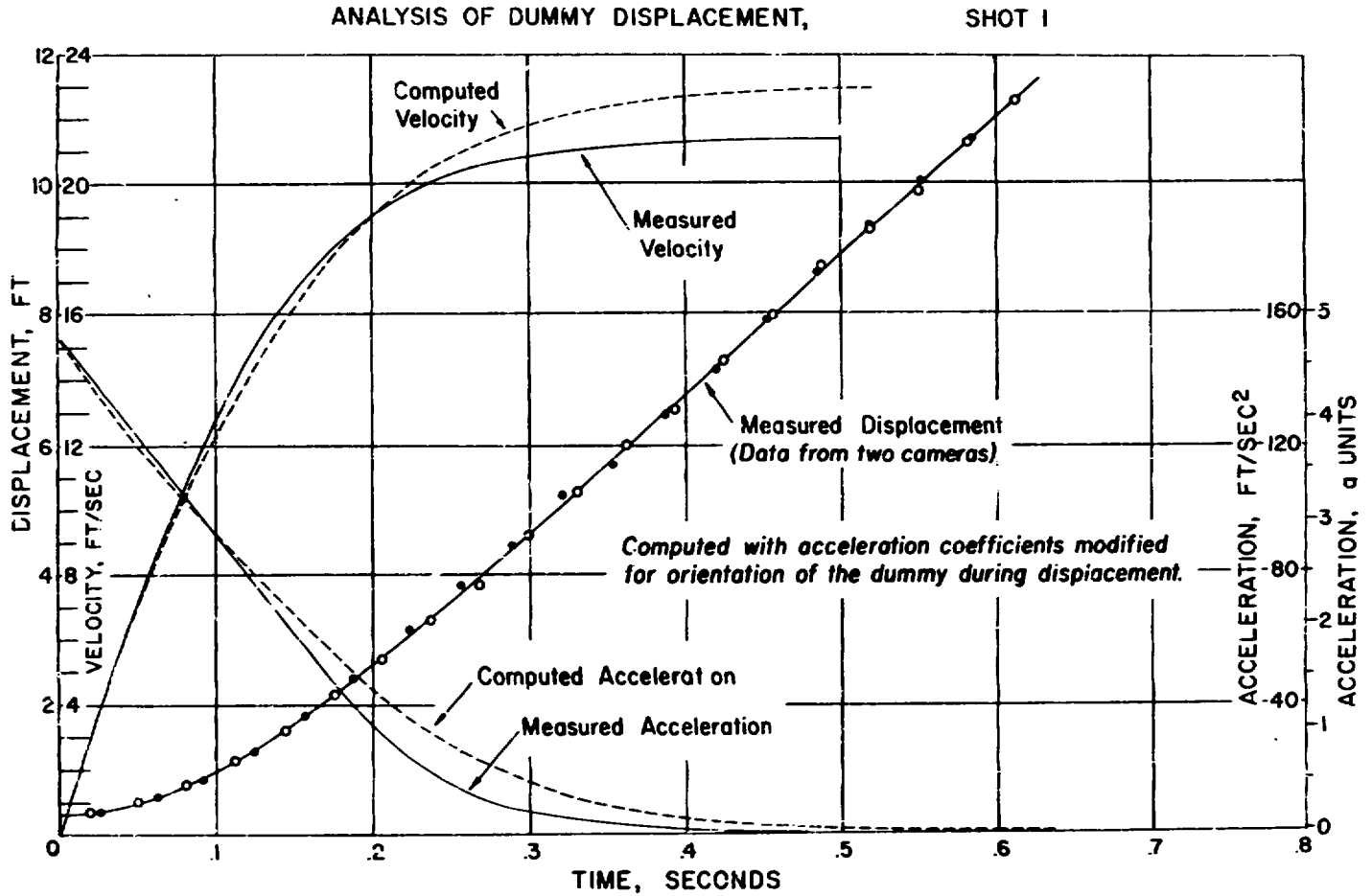

ANALYSIS OF DUMMY DISPLACEMENT, SHOT I
Computed Velocity
Measured Velocity
Measured Displacement (Data from two cameras)
Computed with acceleration coefficients modified for orientation of the dummy during displacement.
Computed Acceleration
Measured Acceleration
DISPLACEMENT, FT
VELOCITY, FT/SEC
ACCELERATION, FT/SEC²
ACCELERATION, g UNITS
TIME, SECONDS
0
.1
.2
.3
.4
.5
.6
.7
.8
12
24
10
20
8
16
6
12
4
8
2
4
160
5
120
4
80
3
40
2
1
0

Fig. 3

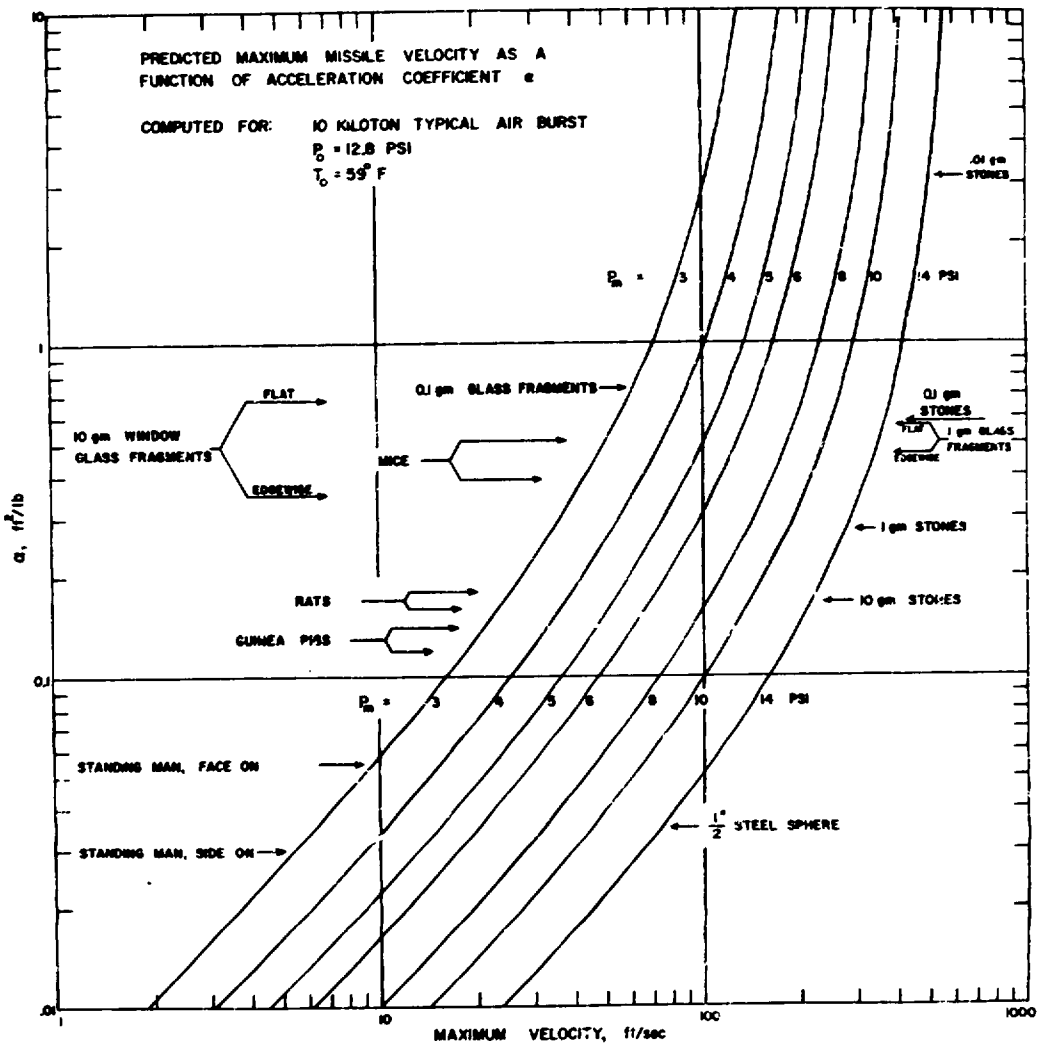

PREDICTED MAXIMUM MISSILE VELOCITY AS A FUNCTION OF ACCELERATION COEFFICIENT α
COMPUTED FOR: 10 KILOTON TYPICAL AIR BURST
P_o = 12.8 PSI
T_o = 59° F
P_m = 3 4 5 6 8 10 14 PSI
.01 gm STONES
0.1 gm GLASS FRAGMENTS
0.1 gm STONES
1 gm GLASS FRAGMENTS
1 gm STONES
10 gm STONES
10 gm WINDOW GLASS FRAGMENTS
FLAT
EDGEWISE
MICE
RATS
GUINEA PIGS
STANDING MAN, FACE ON
STANDING MAN, SIDE ON
½" STEEL SPHERE
α, ft²/lb
MAXIMUM VELOCITY, ft/sec
10
1
0.1
.01
1
10
100
1000

Fig. 4

shown on the left-hand scale. Indicated in the figure are the data for stone and glass missiles of different weight – for mice, rats, guinea pigs, and man oriented face-on and side-on. It is of interest to note that a 1/2-inch steel sphere has about the same alpha as averaged or randomized for man; namely, 0.03 square foot per pound.

Figure 4 also contains iso-overpressure lines for a 10-kiloton air burst at Nevada altitude and a maximum velocity scale computed using the Bowen model[12]. This is shown only to illustrate that for specified blast parameters, providing the applicable alpha is known, one can indeed predict not only maximum velocity for missiles and man, but velocity at various distances of travel.

For example, Table 2 indicates the overpressures required, as well as the range and areas covered, for a 165-pound man to reach a velocity of 10 feet per second if the displacement of interest is 1, 2, 5, and 10 feet, and if the overpressures are due to 1- and 10-megaton surface bursts[2, 12]. Note that the overpressures for the 2 yields are similar for the shorter distances of travel, but become different at 5 and 10 feet. A surprising result of such calculations is the fact that the minimum overpressure at which a velocity of 10 feet per second will occur for the 1- and 10-megaton yields is 1.9 and 1.3 psi, respectively. The corresponding displacement distances at which this velocity is to be anticipated is 28 feet for the 1-megaton yield and 58 feet for the 10-megaton yield.

Such studies as these just described have allowed scaling laws to be developed which will appear in the 1962 edition of The Effects of Nuclear Weapons whereby one may predict the maximum velocity at 10 feet of travel for the "average" man weighing 165 pounds, and for glass fragments between 0.1 and 10 grams in weight, since the latter have nearly the same alpha[15]. The constraint of 10 feet of travel was arbitrarily placed upon distance of travel because this was thought to be applicable to the average home. However, the model, as noted in the previous figure, may be solved for any distance of travel up to the occurrence of maximum velocity[12]. Velocity ranges for man were fixed from 10 to 40

TABLE 2

OVERPRESSURES COMPUTED FOR AN EXPECTED DISPLACEMENT VELOCITY OF 10 FT/SEC FOR A 160 LB. HUMAN BEING TRAVELLING THE INDICATED DISTANCES. RANGES AND AREAS COMPUTED FOR SURFACE BURSTS AT SEA LEVEL

Displacement in ft corresponding to velocity of 10 ft/sec	Incident pressure and corresponding range and area					
	1 MT			10 MT		
	psi	mi	sq mi	psi	mi	sq mi
1	4.3	3.1	30	4.3	6.8	150
2	3.3	3.7	43	3.3	8.0	200
5	2.4	4.7	69	2.3	10	310
10	2.1	5.1	82	1.8	12	450
	1.9*	5.5	95	1.3*	16	800

*Minimum overpressure where velocity of 10 ft/sec is predicted.
Corresponding displacements are: 1 MT - 28 ft
10 MT - 58 ft

feet per second and for glass missiles from 50 to 400 feet per second. The reason for the choices of these velocities will become clear later.

F. Miscellaneous - A few examples from field work will now be given to illustrate some of the physical points just mentioned.

Figure 5[1] shows at the bottom a "free-field" pressure-time curve of classical form recorded 4700 feet from about a 30-kiloton tower burst in Nevada. The incident maximum pressure was 5 psi and the positive-pulse duration almost a second.

The top curve was measured in a concrete bathroom inside a house at the same range with blast shutters over the windows and a wooden blast door closing the entrance. The maximum pressure was about 1.5 psi enduring for almost 1.5 seconds and the rate of pressure rise was quite slow, reaching a maximum pressure in near 500 milliseconds[1].

The two middle curves were from gages placed inside wooden lean-to and corner-room shelters located in the basement of a house also at 4700 feet from ground zero. There was some degrading of the pressure rise, a slight decrease in the maximal overpressure, and no change in the duration[1].

No damage was noted to animals in these shelters, even though the houses were completely destroyed.

Figure 6 shows pressure-time curves recorded in the 1953 test series inside 48-feet-long, 7-feet-diameter tubular structures without doors subjected up to 15 psi "free-field" incident overpressures[1]. The shelters were entered by walking down ramps, turning right and sharp right again to gain access to the main chambers. Note the top curve from one structure rising in 2 steps to a maximum of about 25 psi. The wall gages recorded the initial pressure pulse which entered the shelter and then later the pressure reflected from the far end of the structure.

Dogs restrained to avoid displacement suffered no fatalities, but a few exhibited ataxia; too, lung hemorrhage was severe in some[1]. This was surprising since 75 psi produced by detonation of 4400 pounds

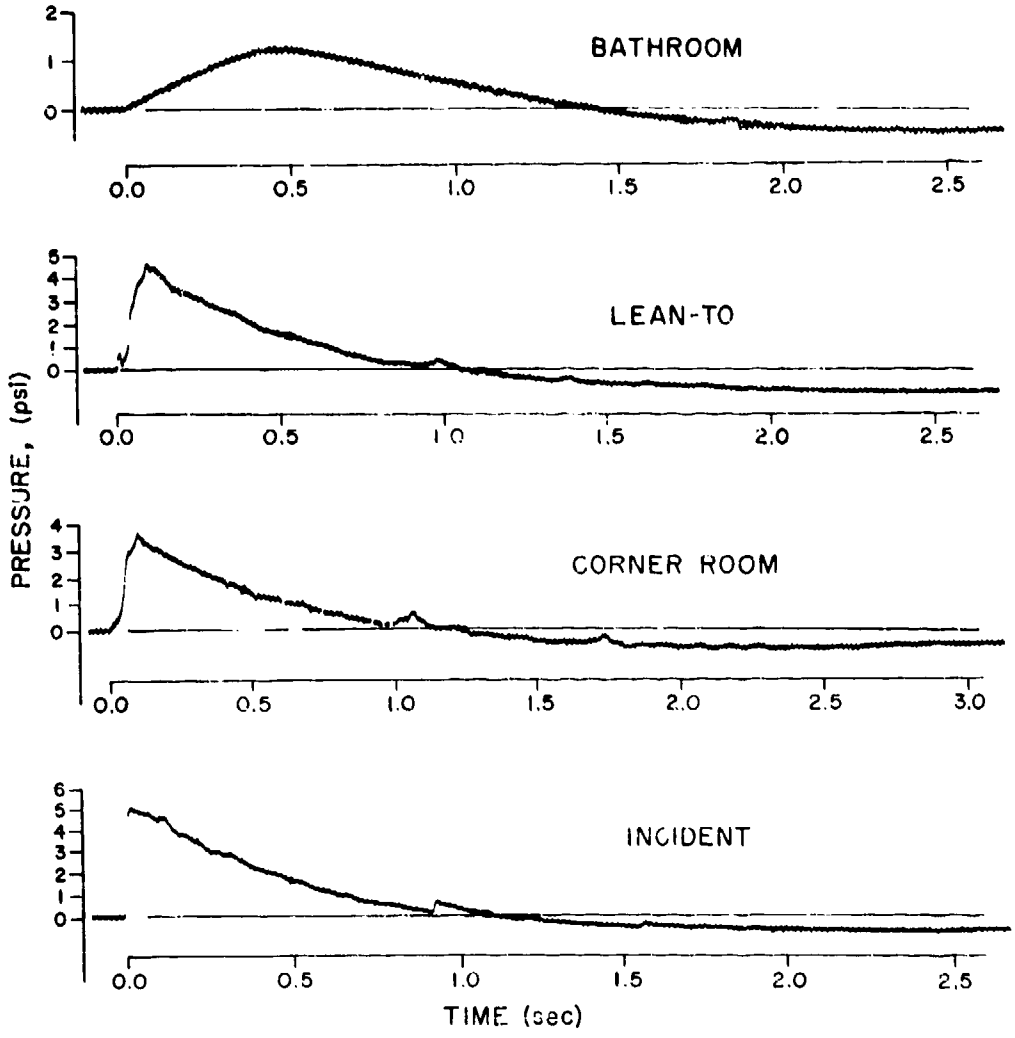

BATHROOM
LEAN-TO
CORNER ROOM
INCIDENT
PRESSURE, (psi)
TIME (sec)
0.0
0.5
1.0
1.5
2.0
2.5
3.0

Fig. 5

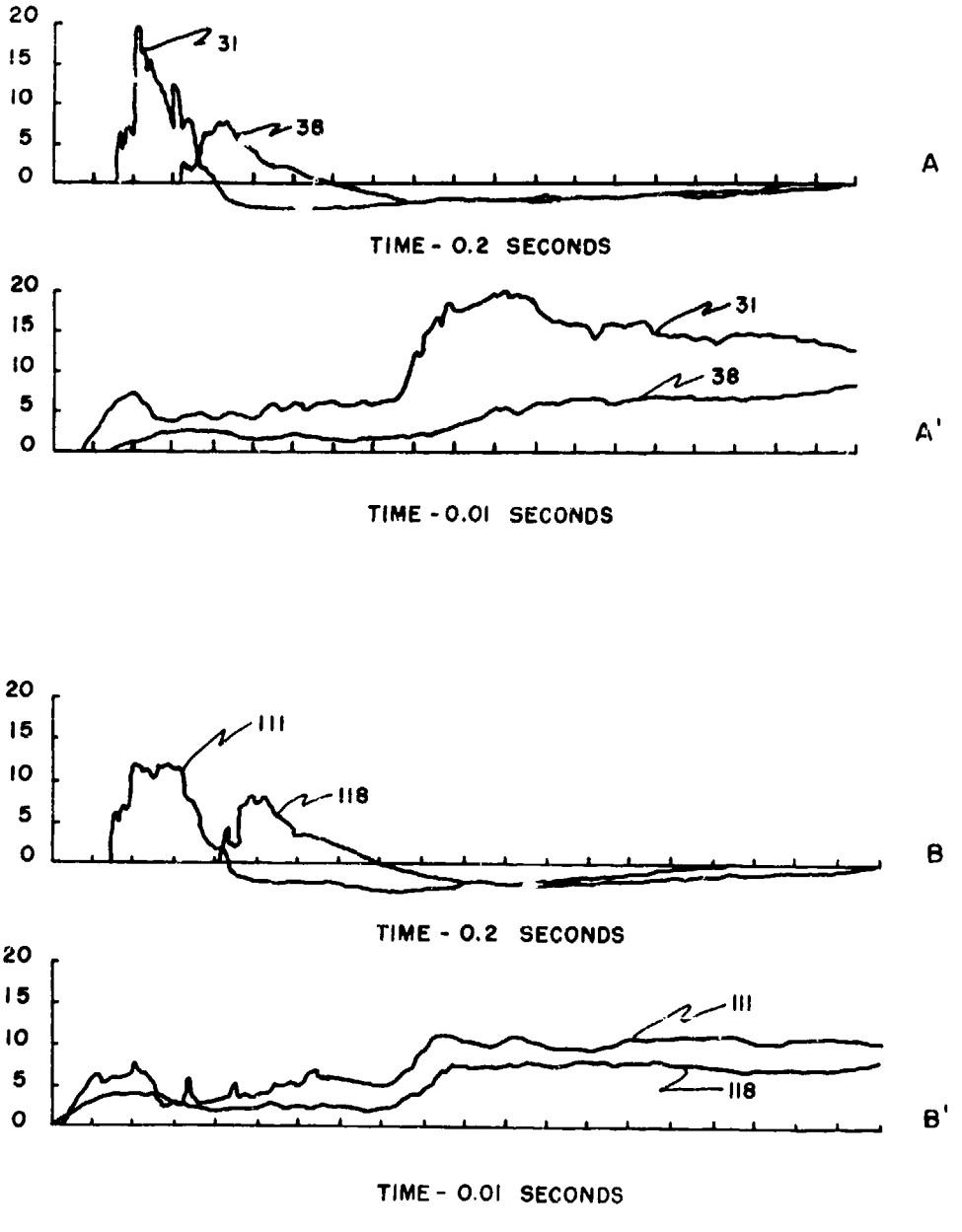

20
15
10
5
0
31
38
A
TIME - 0.2 SECONDS
20
15
10
5
0
31
38
A'
TIME - 0.01 SECONDS
20
15
10
5
0
111
118
B
TIME - 0.2 SECONDS
20
15
10
5
0
111
118
B'
TIME - 0.01 SECONDS

Fig. 6

of high explosive was the lowest overpressure then known to fatally injure the canine species[9].

Consider the lower curve in Figure 7 showing a maximum pressure of 85 psi reached in about 5 milliseconds recorded inside a buried basement exit shelter subjected to only 40 psi incident "free-field" overpressure[1]. The shelter was entered by a 2-feet-wide, steep stairway opening to the left into a chamber about 12 feet long, 3 feet wide and 5 feet high.

Note the reverberations of pressure in another shelter of the same type due to multiple reflections from the ends of the structure (the curve shown second from the bottom). Dogs were recovered alive, though singed, from such structures.

Figure 8 shows the plan of a buried structure tested open in 1955[1] and subjected to a "free-field" incident overpressure of close to 90 psi at a range of 1050 feet, about 300 feet outside the fireball. The yield was approximately 30 kilotons. Each chamber, instrumented by wall gages for pressure and temperature, was 12 x 12 feet square and 8 feet high. One, the "fast-fill" side, filled with pressure through the main entryway, and the other, the "slow-fill" chamber, through a 3-feet-square escape hatch. Animals were placed on the benches and in cages suspended from the ceiling. Note the position just in front of the main entryway where a dog was located with a Q-gage near by; this will be referred to later.

Figure 9 shows at the top the pressure-time curve in the "slow-fill" chamber[1]. Maximum pressure was about 22 psi, rising to a maximum in about 120 milliseconds and enduring for near 500 milliseconds. Some small animals suffered fatality and the winds made quite a shambles of the contents of the room. Too, some animals were singed. Air temperatures near the walls peaked to about 360° C[16]. No dogs were injured except for loss of some eardrums.

The second curve, rising slowly to a maximum pressure of 66 psi in 90 milliseconds, and enduring about 350 milliseconds, was recorded inside the "fast-fill" chamber.

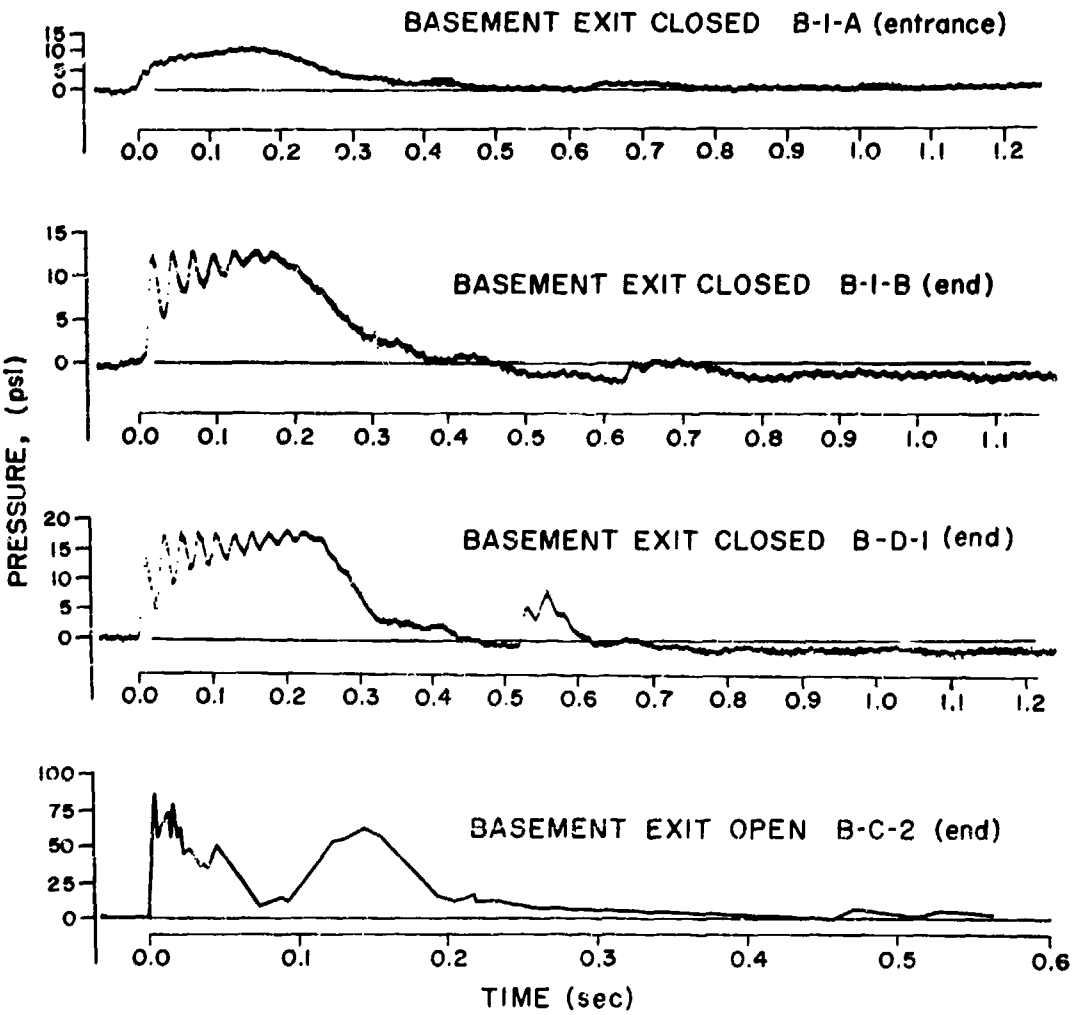

BASEMENT EXIT CLOSED B-I-A (entrance)
BASEMENT EXIT CLOSED B-I-B (end)
BASEMENT EXIT CLOSED B-D-I (end)
BASEMENT EXIT OPEN B-C-2 (end)
PRESSURE, (psi)
TIME (sec)

Fig. 7

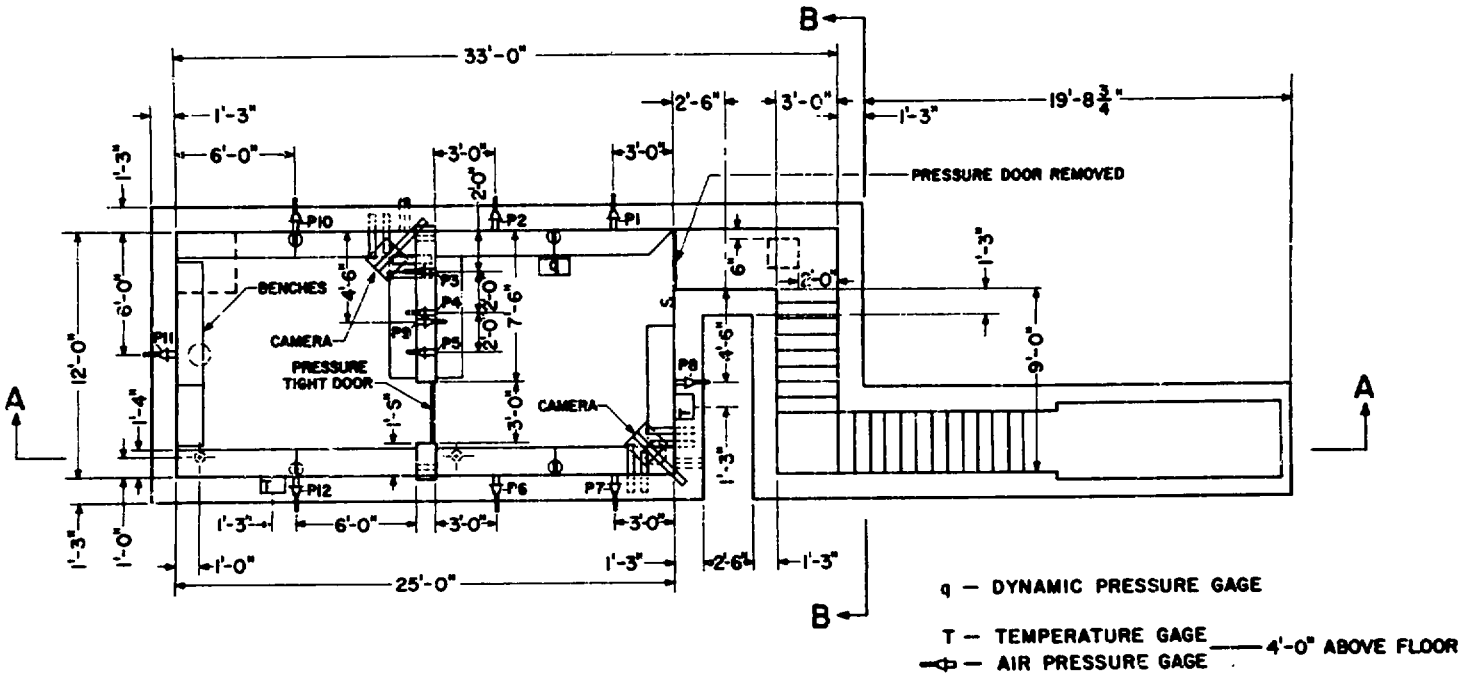

B
33'-0"
1'-3"
6'-0"
3'-0"
3'-0"
2'-6"
3'-0"
1'-3"
19'-8¾"
PRESSURE DOOR REMOVED
P10
P2
P1
P3
P4
P5
P11
BENCHES
CAMERA
PRESSURE TIGHT DOOR
P8
CAMERA
A
A
12'-0"
6'-0"
1'-4"
P12
P6
P7
1'-3"
1'-0"
1'-3"
6'-0"
3'-0"
3'-0"
1'-0"
1'-3"
2'-6"
1'-3"
25'-0"
B
q — DYNAMIC PRESSURE GAGE
T — TEMPERATURE GAGE
AIR PRESSURE GAGE
4'-0" ABOVE FLOOR

Fig. 8

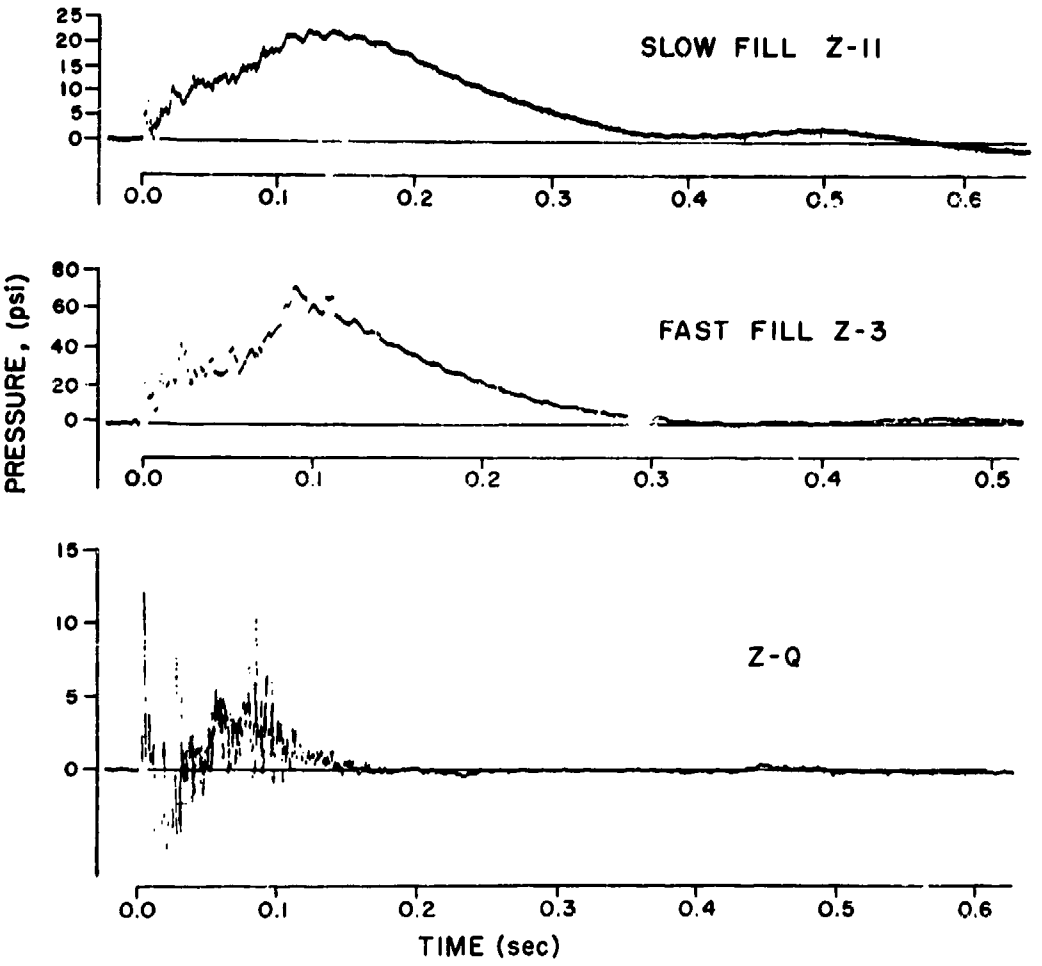

SLOW FILL Z-11
FAST FILL Z-3
Z-Q
PRESSURE, (psi)
TIME (sec)
25
20
15
10
5
0
80
60
40
20
0
0.0
0.1
0.2
0.3
0.4
0.5
0.6

Fig. 9

The lower curve is that of the Q-gage. This indicated a maximum Q of about 12 psi.

The black-furred dog just in front of the door, in spite of stout restraints, was hurled violently against the far wall leaving the imprint shown in Figure 10[1]. The dark area is carbon from the animal's singed fur. The animal was killed instantly from impact and also suffered skin burns. All ther animals were recovered alive, though one suffered pneumothorax; most were singed moderately on the side toward the room where the hot-gas winds made most contact; the majority of the eardrums were ruptured. The air temperatures near the walls peaked to about 320°C[16]; outside the structure a thermal flux of about 600-700 cal/cm^2 occurred[6].

Figure 11 shows the postshot state of the harness of the displaced animal and Figure 12 one of the strong steel harness snaps which was sheared as it might have been if firmly placed in a vice and hit sharply with a heavy hammer[1].

These last few figures were shown to emphasize three points; namely,

1. That what occurs inside a structure, depending upon its design, may or may not be indicated by the anticipated "free-field" overpressures;

2. That wind inside a structure may, because of its displacement potential and in the absence of effective restraints or solid deflecting baffles, be far more hazardous than the overpressure itself; and

3. That mammals, given restraints plus minimal but appropriate shielding from "free-field" pressures and winds, have indeed survived in surprisingly high-overpressure regions and well inside those ranges which completely destroy houses and fairly heavy above-ground structures.

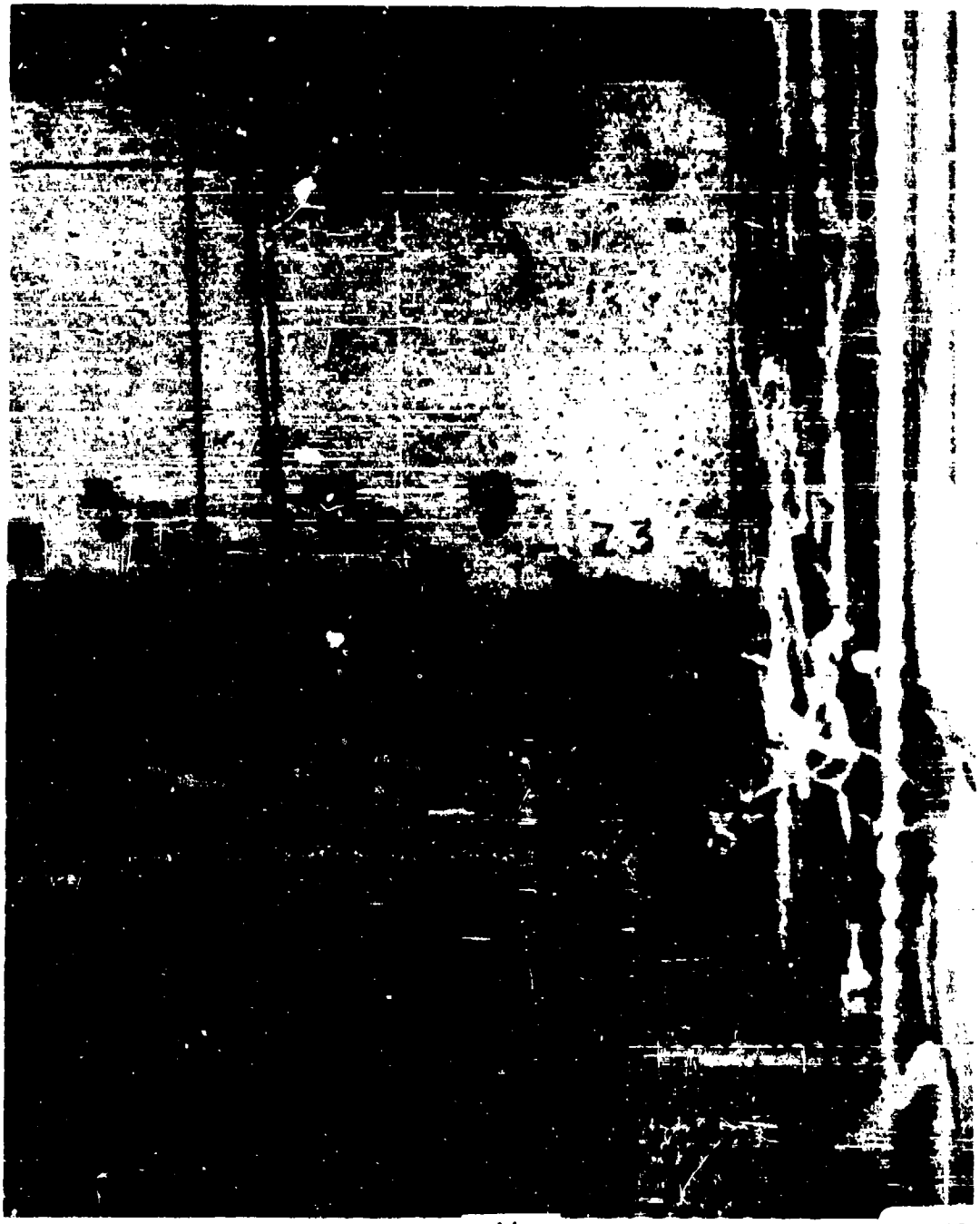


Fig. 10

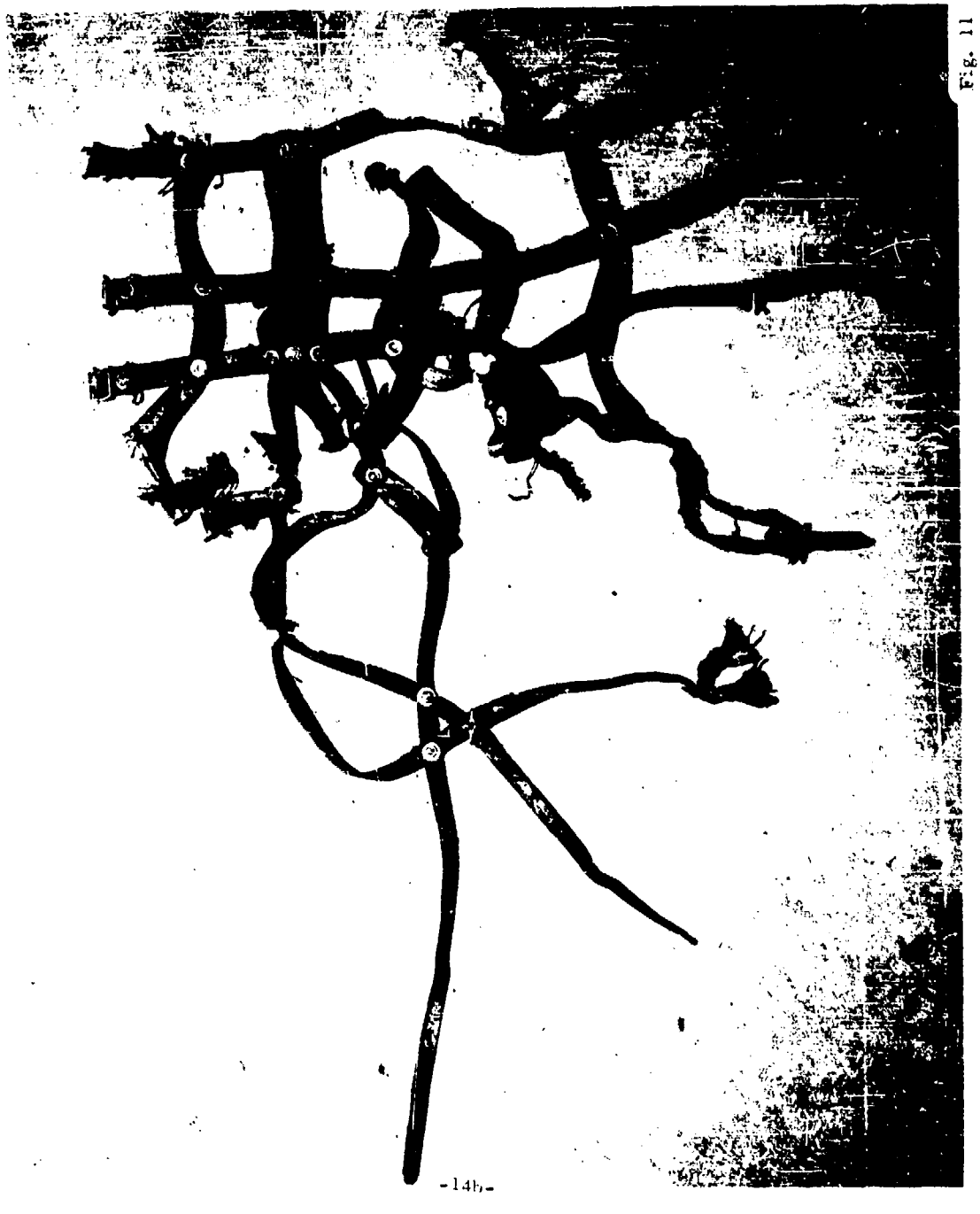


Fig. 11

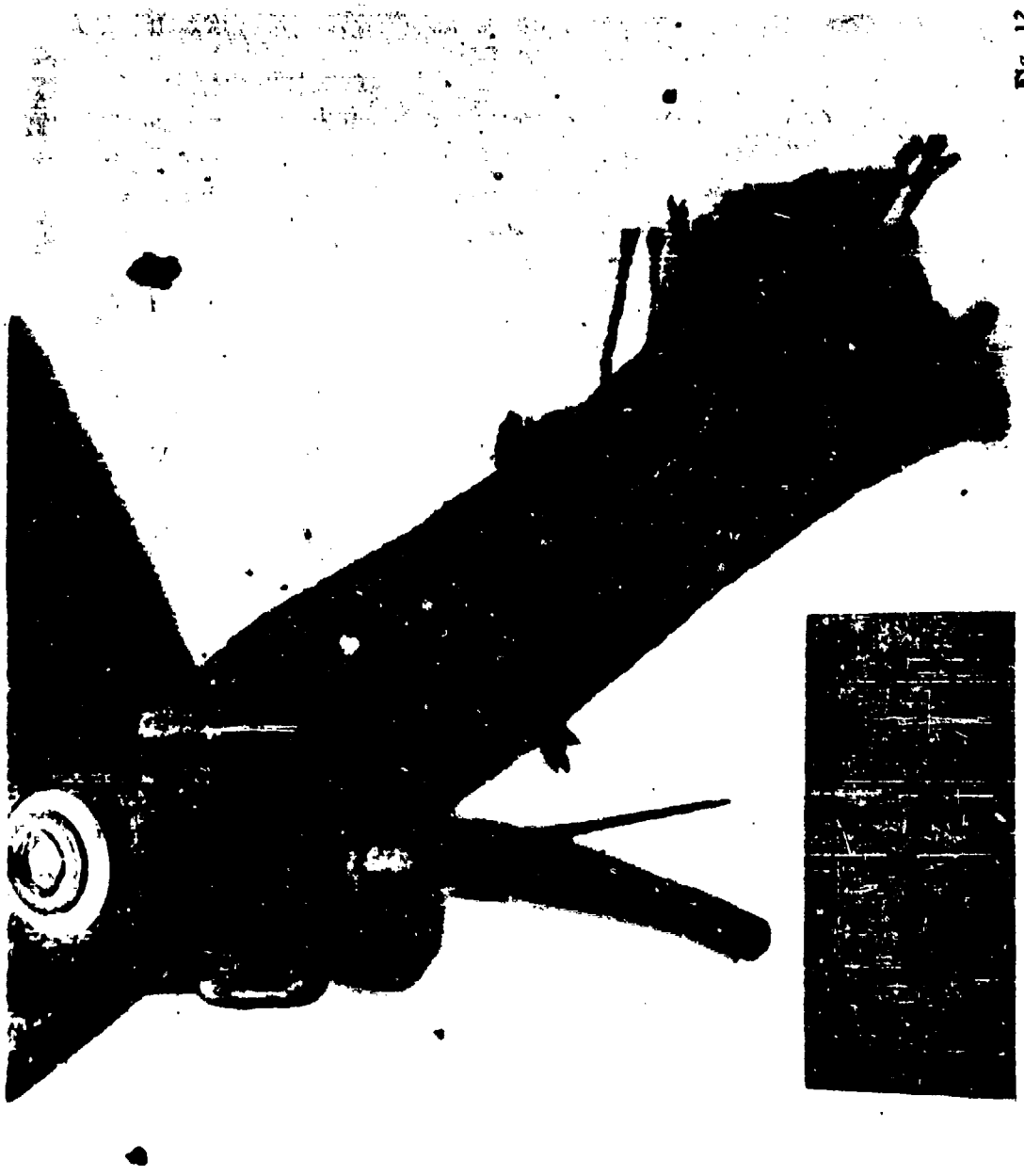


Fig. 12

G. Spalling and Implosion Effects - There are two known physical phenomena that are germane to understanding the biological blast hazard. The first of these has been called the "spalling effect" by Schardin and can be illustrated by noting Figure 13 which shows a pressure pulse in a circular glass plate induced by a small explosive detonated in a centrally-drilled hole[8]. The "shock front" travels radially to the periphery at which time it "attempts" to pass from the dense glass to the less-dense air. The result is a negative reflection and the entire periphery of the plate shatters before the breaking cracks directly induced by the explosion can reach the edge[8].

The second phenomena, termed the "implosion effect" is of considerable interest and concerns the chain of events which follows a small detonation in water through which air bubbles are rising. When the induced shock shown in the upper left of Figure 14, also from Schardin[8], reaches the bubbles, each one behaves as though it were an explosive source as shown in the remaining consecutive pictures of the figure. Apparently, spalling occurs at the air-water-interface and the air volume is decreased accordingly, tending to raise the pressure and temperature inside the bubble. Each bubble is compressible and the particle velocity of the water also tends to decrease the gas volume which, in turn, increases the pressure and temperature inside the air phase. The result is the development of very high pressures and temperatures and perhaps the production of steam. Whatever the factors at play, the result is a violent disturbance in the vicinity of the originally-stable air bubble.

Schardine[8] and others[1, 9, 17] have suggested that shock disturbance external to the body may induce an internal pressure pulse in the fluid phase and that spalling and implosion phenomena may become "active" at the junction of tissues of different densities. At least, these physical facts alert one to possible internal events which may be important in the etiology of blast damage and bring up the problem area of biophysical interaction mentioned earlier (see Table 1).

Figure 13 .—Two spark-kinematographic pictures of the propagation of a shock wave in a circular glass plate. Spalling effect.

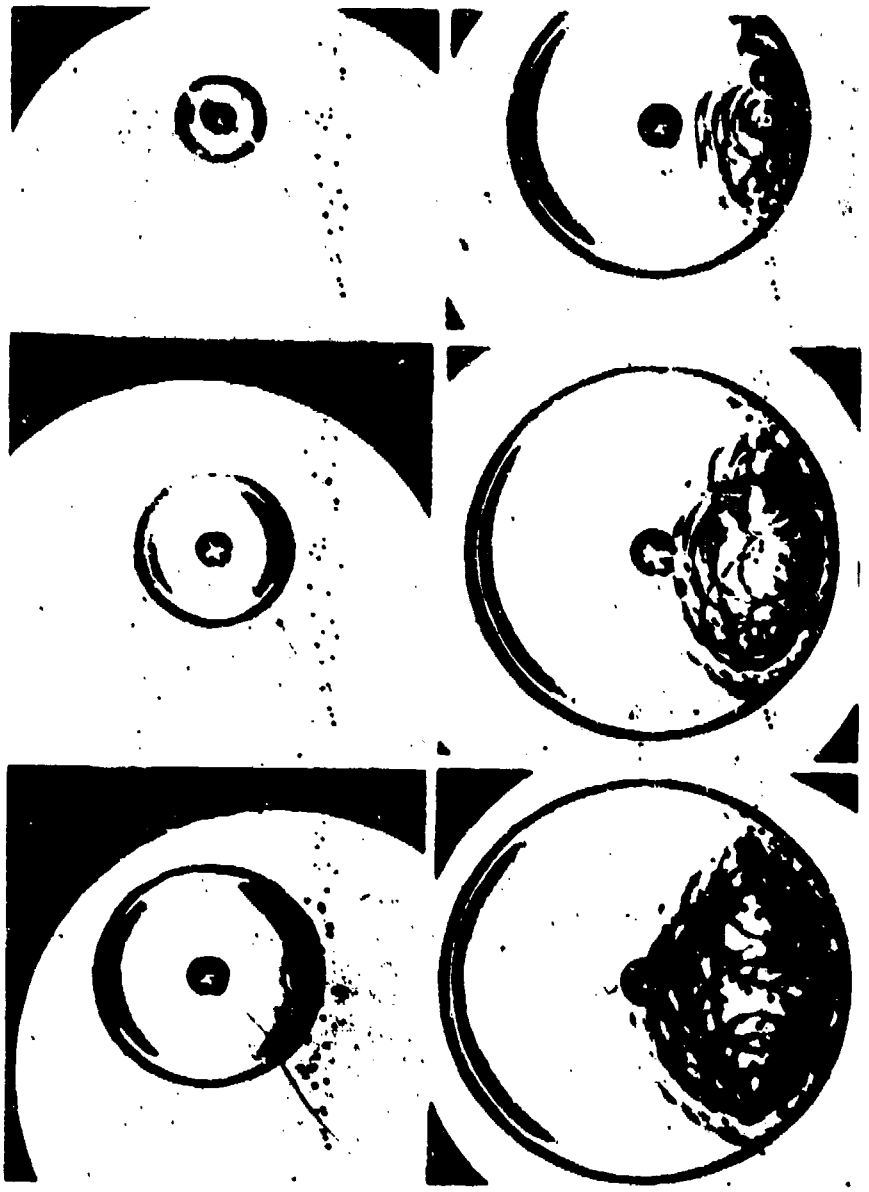


Figure 14. Six consecutive phases of an underwater bo... passing over enclosed air bubbles. Implosion of the air bubb...

H. Biophysical Considerations - Early German[8,9,17,19] and English[20-23] experiences are of qualitative interest in approaching the biophysics of blast damage from overpressure. Let us consider Diagram No. 4 in Figure 15 from Benzinger[17] and visualize a dog enclosed except for his head in a rigid metal box and imagine detonation of a charge near by which would be fatal if the box were not shielding the animal. Nothing happens to the animal following the explosion. The impact of the shock with the head in the open is not fatal as is also the case for the dog exposed to underwater blast with the head immersed as in Diagram No. 2.

If a tracheotomy tube as in Diagram No. 5 is employed, the animal also survives, indicating that pressure traveling into the lungs from a small charge creating a "short"-duration pulse is not harmful.

The animal in Diagram No. 3 with a tracheotomy tube and funnel with a gas mask filter to impede air flow, dies just as readily as without the tracheotomy tube. These facts indicate that the impact of the shock overpressure with the trunk of the animal is critical. A similar result is obtained with a dog immersed to the neck as shown in Diagram No. 1 of Figure 15. In the latter case, pathology in both the abdomen and chest occurs, and signs of nervous system damage are also observed. The animal is likely to die quite quickly.

If, however, the animal is immersed hind legs first only to the diaphragm, pathology occurs in the abdomen and not the chest. No damage to the central nervous system is seen and if fatality ensues, it is not acute and is rather a delayed matter due to the sequellae of injury to the abdominal organs. This experiment focuses attention on the chest as one critical organ, damage to which is responsible for early lethality[17].

Use of a thin, but rigid, plaster cast over the trunk to prevent overexpansion of the lungs during the underpressure phase of the pressure pulse gives the animal no protection. However, a unilateral pneumothorax does protect the ipsilateral lung compared with the contralateral one[9,17].

Thus, it is well to consider the possible events which involve the

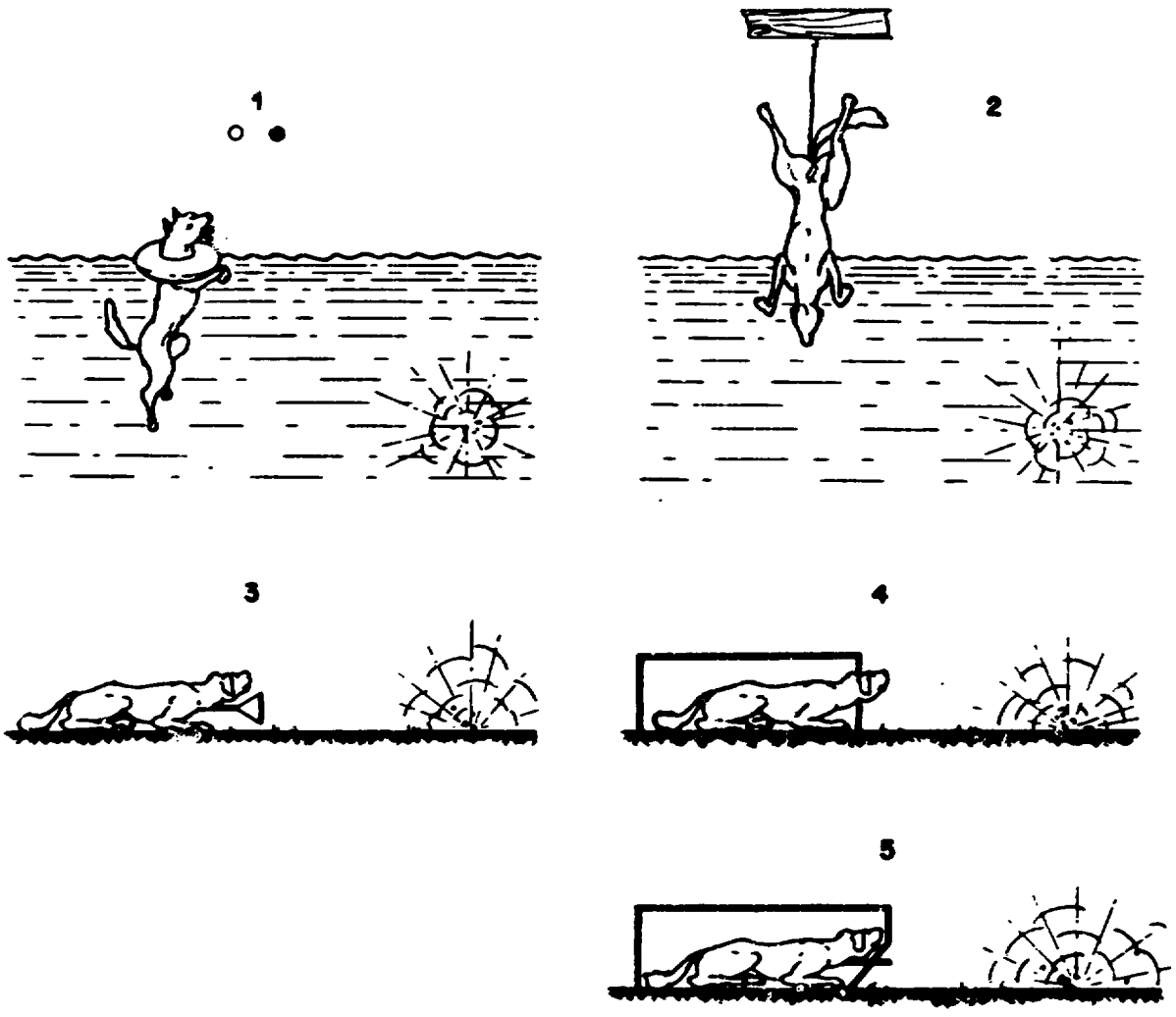


Fig. 15

thorax, including the abdomen which is "coupled" to the chest through the diaphragm.

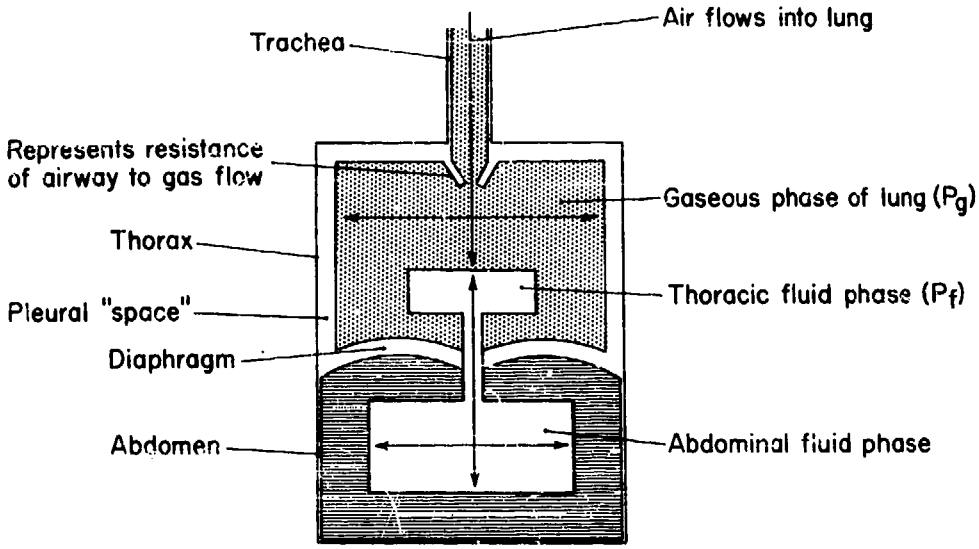
To accomplish this, let us leave the soul to the local vicar, assign the personality to the psychiatrist, and view man as a rather simple model consisting of fluid within the skin, complicated by the inclusion of tissue of different density from hard bone to the spongy lung.

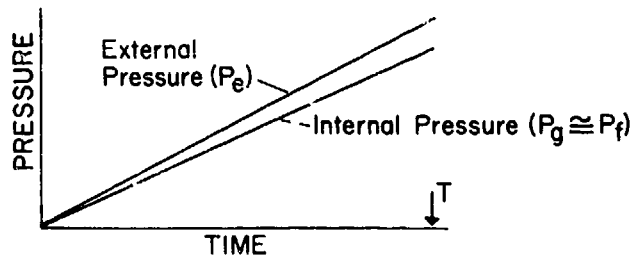
Figure 16 will be of aid[3]. The thorax and abdomen are shown as boxes above and below separated by the diaphragm. A liquid and air phase is imagined in the thorax with the former coupled through the vascular system with a fluid compartment in the abdomen. A constriction is shown at the lower end of the trachea to represent not so much the resistance to air flow in the larynx, but that due to the small caliber of the tiny respiratory bronchioles leading to the alveoli.

If one imagines a slow enough rise in environmental pressure as shown in the diagram below the model, there will be no significant differences between the external pressure and that existing inside the body. This is so because there is time for air to flow through the airways and maintain near equilibrium with the outside pressure.

In Figure 17 a more rapid, but moderate, rise in environmental pr ssure is depicted[3]. The internal fluid pressure follows the external pressure fairly faithfully, but since air flow into the chest is relatively slow, the internal gaseous pressure remains negative to the external pressure. This allows the latter to push in the chest and abdominal walls, elevating the diaphragm, a process which decreases the volume of the gas in the chest. Such events along with some air flow and some fluid flow into the thorax may bring equilibrium between the internal and external pressures at some finite time, T, as shown in the diagram.

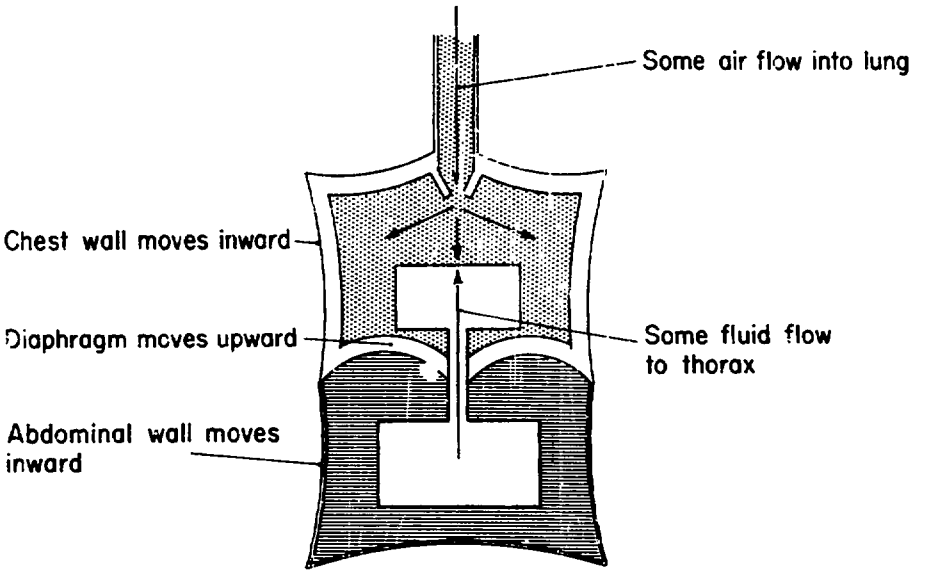
Now visualize the development of a yet more rapid and fairly high rise in external pressure of long duration as shown in Figure 18[3]. Maximal implosion of the chest and abdominal walls will occur but of insufficient amount to bring the internal gas pressure even close to the ambient. The result will be a powerful squeeze and a more prolonged fluid flow into the still compressible gas compartment of the thorax. Severe hemorrhage as in the squeeze syndrome in divers is very likely to occur.

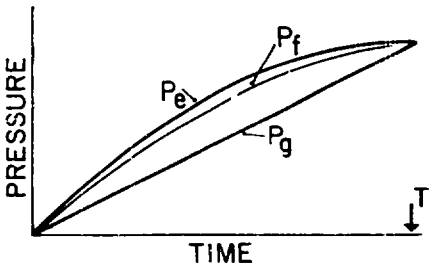




$P_e \cong P_g \cong P_f$

Fig. 16

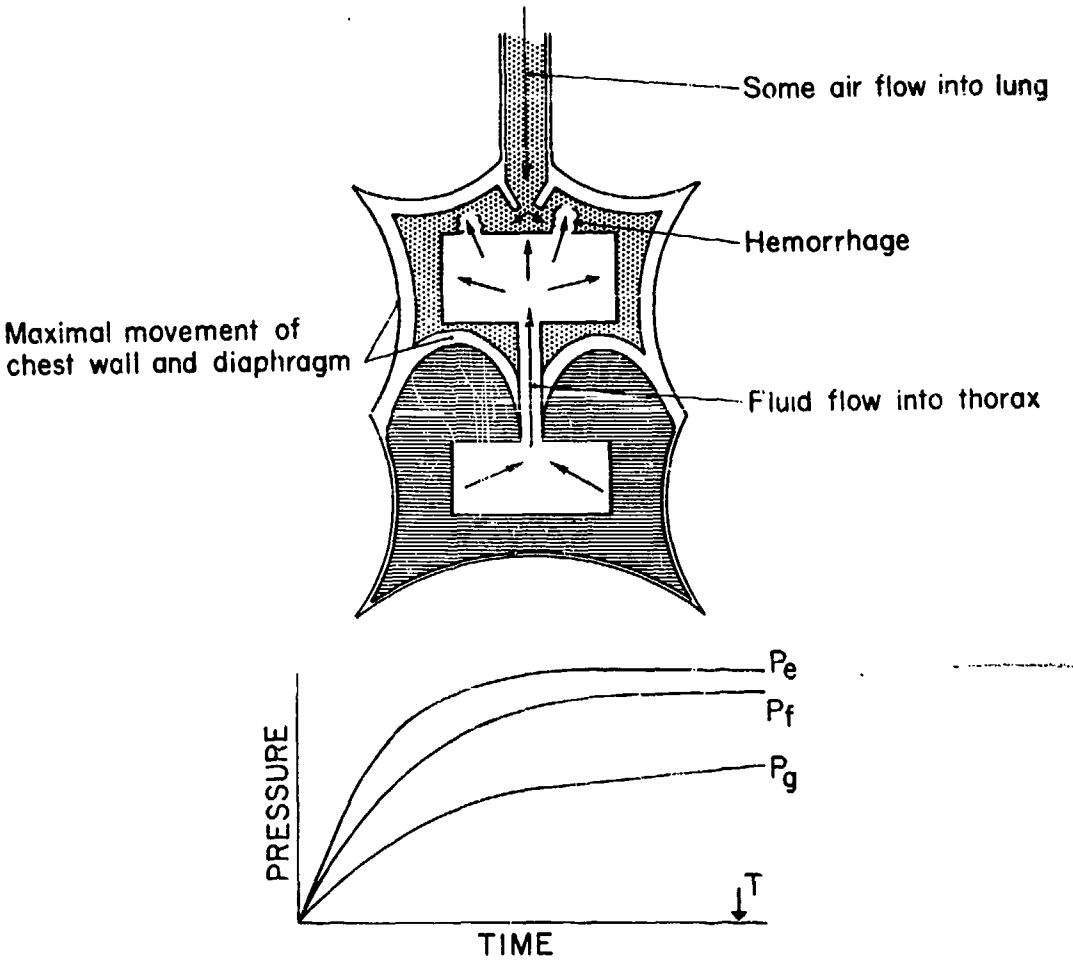




$P_e \cong P_g = P_f$ at time T

$P_e \cong P_f > P_g$ before time T

Fig. 17



$P_e > P_f > P_g$
before and up to time T

Fig. 18

Figure 19 depicts the application of an instantaneous rise in pressure of considerable magnitude and duration[3]. There is initially hardly time for gas flow through the airways and, though this plus maximal implosion and fluid flow will occur later, there exists the likelihood of shock pressures moving through the body's fluid media to cause spalling and implosion effects at the air-fluid interfaces and functions of other tissues of different density.

Lastly, contemplate the sudden application of a "fast"-rising, quite "short"-duration pulse of overpressure as diagrammed in Figure 20[3]. There is hardly time for much movement of the chest wall and abdomen since these have considerable mass and are therefore inertia sensitive. Neither is there time for much fluid or air flow. The induced internal pressure pulse moving at near 5500 feet per second through fluid may well reach the air-fluid interface of the lung before the air shock traveling at 1100 feet per second can arrive and provide pressure compensation. Therefore, the first dangerous pressure differential may well be between the high fluid pressures in the lung and the relatively low gas pressures nearby. Rupture of the blood vessels may occur and, if this is complicated by effects due to spalling and implosion events, it is not hard to imagine great damage to the lung with air gaining access to the peripheral circulation to explain the central nervous system signs mentioned a few moments ago.

Consideration of these simple models allows one to postulate that primary blast damage may involve at least three mechanisms; namely, (a) very fast events involving internal fluid-pressure pulses – and indeed these have been directly investigated by Clemedson in Sweden[24] – that cause damage at interfaces of different density; (b) "rapid" inertia-sensitive responses involving the chest wall and diaphragm; and (c) relatively "slow" inertia-sensitive occurrences encompassing fluid flow into the lung and some of the other air-containing organs.

In addition, once the external and internal pressures become equalized from elastic recoil of the system during the maintenance of "long"-

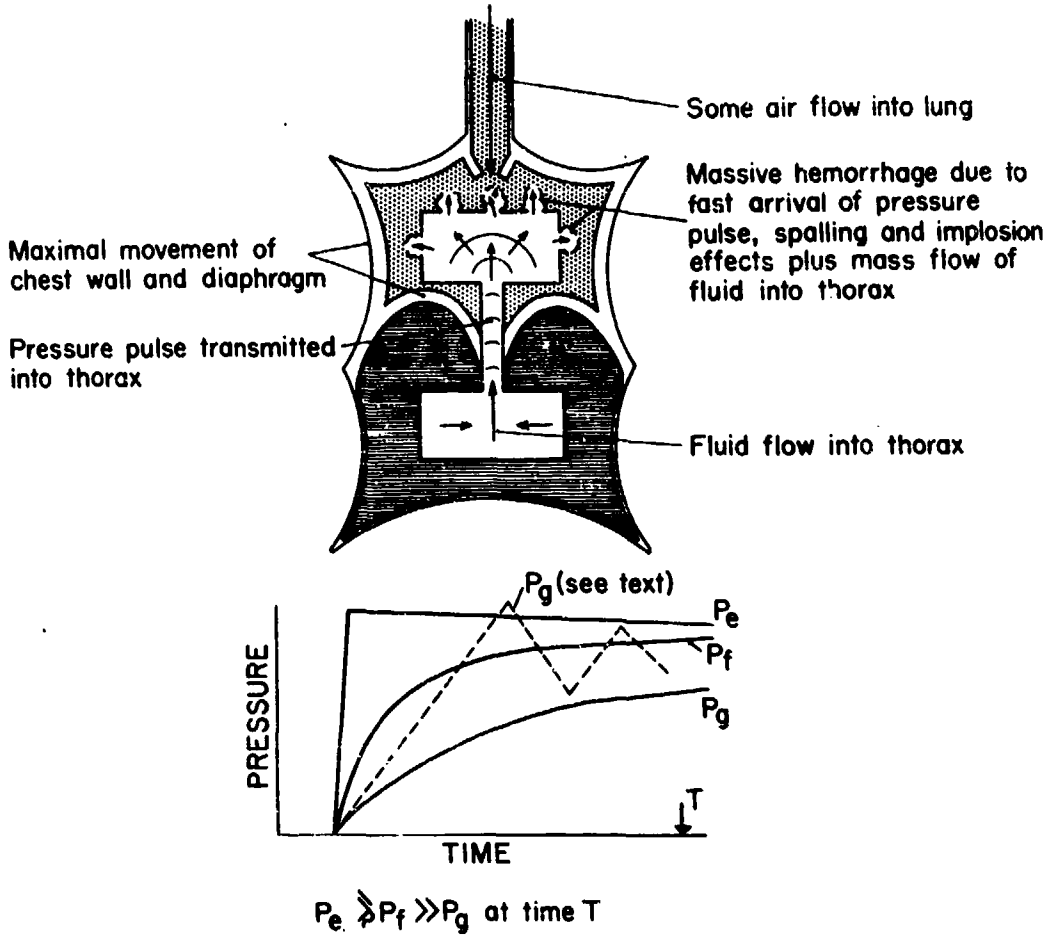

Some air flow into lung
Massive hemorrhage due to fast arrival of pressure pulse, spalling and implosion effects plus mass flow of fluid into thorax
Maximal movement of chest wall and diaphragm
Pressure pulse transmitted into thorax
Fluid flow into thorax
P_g (see text)
P_e
P_f
P_g
PRESSURE
T
TIME
$P_e \geqslant P_f \gg P_g$ at time T

Fig. 19

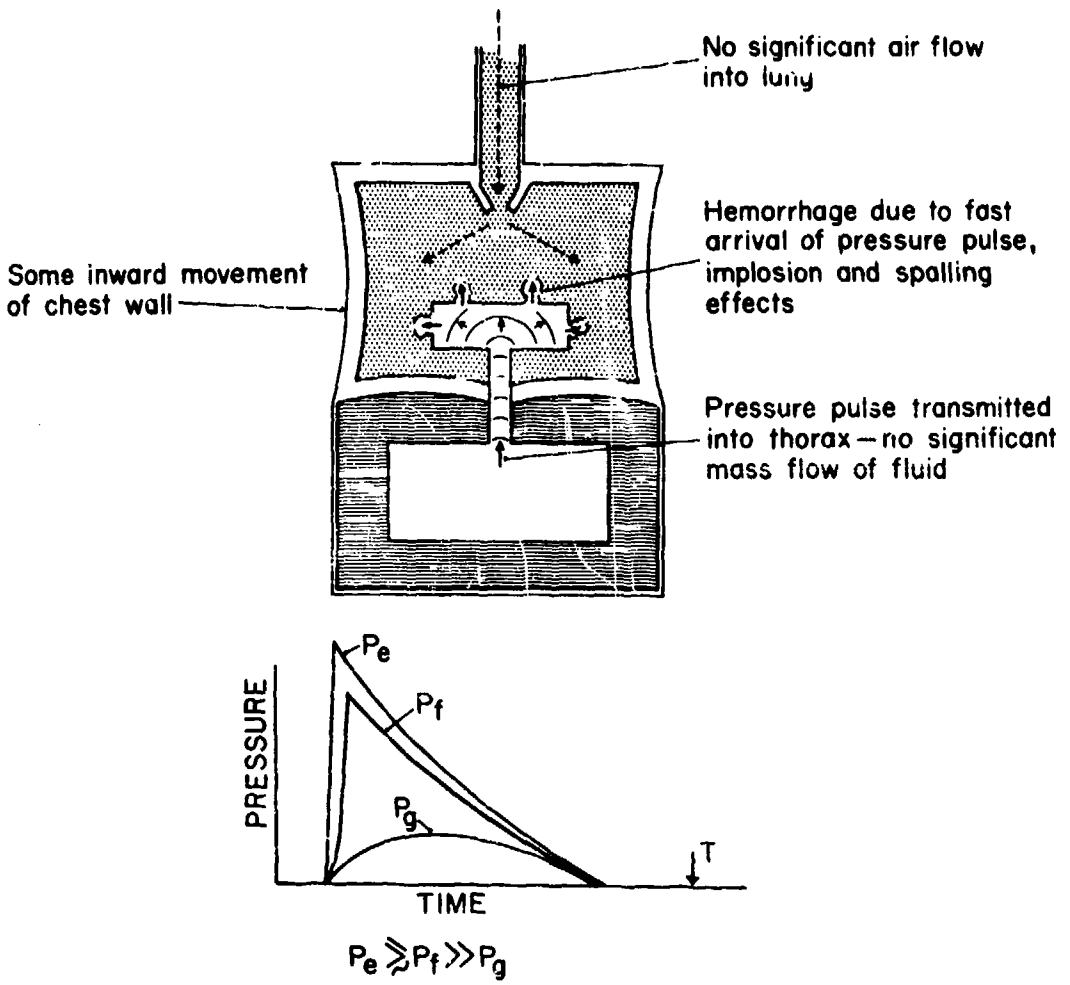

No significant air flow into lung
Hemorrhage due to fast arrival of pressure pulse, implosion and spalling effects
Some inward movement of chest wall
Pressure pulse transmitted into thorax — no significant mass flow of fluid
Pe
Pf
Pg
PRESSURE
T
TIME
Pe ≳ Pf ≫ Pg

Fig. 20

duration overpressures, it is possible that decompression-like pathology might ensue[1, 3]. This, of course, is dependent entirely upon the magnitude and rate of pressure drop, and because the latter is relatively prolonged in the case of blast overpressure, rapid decompression is not likely under ordinary circumstances to be a contributing factor to blast pathology.

IV. Biological Response

The discussion will now be turned to the area of biological response and selected quantitative data of fairly recent origin will be presented to elucidate primary, secondary, and tertiary effects more fully.

A. Primary Effects

1. Single-pulse "Fast"-rising Overpressures of "Long" and "Short" Duration

Considerable data over the past one to two years have been obtained by Dr. Donald Richmond[18, 25-27] using special adaptations of shock-tube technology along with experiments with high explosives, the latter carried out cooperatively with Sandia Corporation personnel. One series of experiments involving six species of animals will now be discussed wherein single-pulse "fast"-rising overpressures of 400 milliseconds were employed[26].

A diagram of the shock tube used to produce the overpressures is shown in Figure 21. A 40-inch diameter, 1000-gallon pressure chamber — the driver section — is shown on the right. This is separated by a diaphragm from the expansion portion of the tube[26]. The latter consists of 30 feet of 24-inch tubing expanded into a test chamber 40 inches in diameter and 22 feet long. Three vents to control duration and to "tailor" the shape of the pressure wave are located at the upstream end of the test chamber. Animals, depending upon their size, are exposed in harness against the plate closing the end of the tube or in individual diamond-mesh steel cages bolted to the end-plate. Fast-responding, piezo-electric instrumentation is located near the animal station.

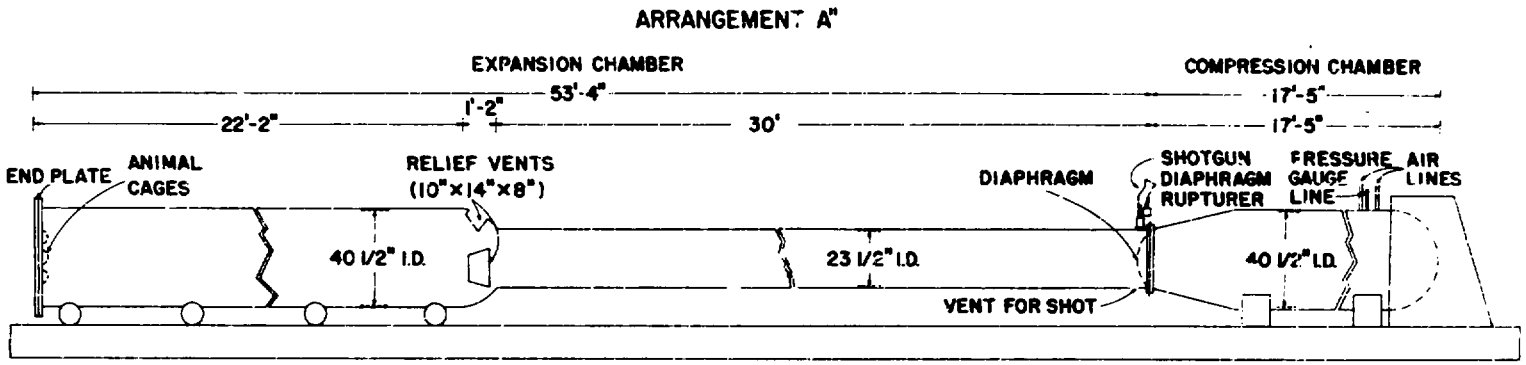

ARRANGEMENT A"
EXPANSION CHAMBER
COMPRESSION CHAMBER
53'-4"
17'-5"
22'-2"
1'-2"
30'
17'-5"
END PLATE
ANIMAL CAGES
RELIEF VENTS (10"×14"×8")
DIAPHRAGM
SHOTGUN DIAPHRAGM RUPTURER
PRESSURE GAUGE LINE
AIR LINES
40 1/2" I.D.
23 1/2" I.D.
40 1/2" I.D.
VENT FOR SHOT

Fig. 21

After pressurization of the driver section, the mylar diaphragm is ruptured by shot from a sawed-off shotgun. Air rushes downstream, "shocks-up" and then reflects from the end-plate. The animals in this geometry "see" the incident wave and, almost instantaneously, the reflected pressure.

Figure 22 shows records of the overpressures achieved[26] The upper left tracing was taken with a gage mounted face-on to the end-plate. Each vertical line represents 200 microseconds. That the rate of pressure rise is very rapid is apparent also from the trace at the upper right for which each vertical line represents 100 microseconds. The multiple oscillations are those from the natural periods of the gages which are in the order of 20,000 to 40,000 cycles per second.

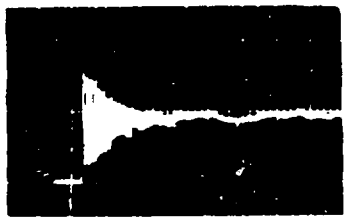
The lower left record was taken with a gage mounted in the wall of the test section 3 inches from the end-plate. The record faithfully shows first the incident wave, a pause, and then the return of the reflected pressure.

The lower right record, run quite slowly — each vertical line being equal to 50 milliseconds — shows that the over-all wave is nearly ideal or classical, though a few oscillations of pressure occur during the falling phase of the pulse.

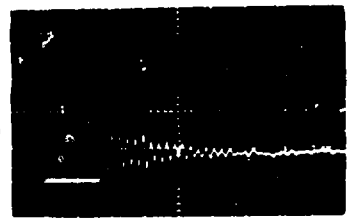
A total of 569 animals — 140 mice, 164 rats, 96 guinea pigs, 104 rabbits, 35 dogs and 30 goats — were systematically exposed and 24-hour mortality curves were obtained. These were linearized by the probit technique of Finney[28] and the reflected shock overpressures associated with 50 per cent mortality were calculated for each species. The mortality curves are shown in Figure 23 as are the P_{50} figures[26] The latter were about 31, 36, 35, 30, 48, and 53 for the mouse, rat, guinea pig, rabbit, dog, and goat, respectively.

Figure 24 depicts the P_{50} values for each species as a function of average body weight and shows the regression line obtained by the least-squares fit[26]. The equation has a standard error of 0.06 log units or about 14 per cent.

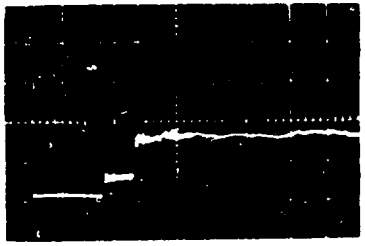
PRESSURE-TIME RECORDS
QUARTZ PIEZO GAUGES



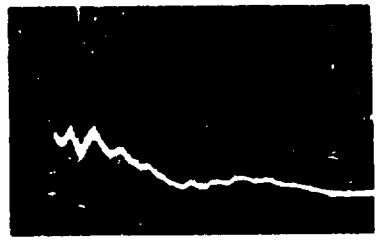
0.2 msec/cm
Gauge 1 Face-on at End Plate



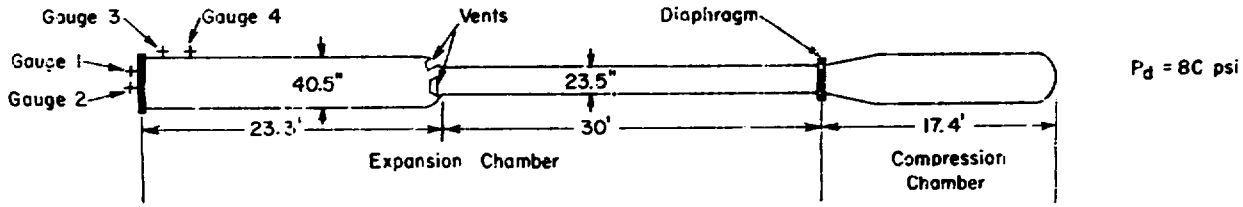
0.1 msec/cm
Gauge 2 Face-on at End Plate



0.5 msec/cm
Gauge 3 Side-on 3" from End Plate



50 msec/cm
Gauge 4 Side-on 9" from End Plate



P_d = 80 psi

Arrangement A"

Fig. 22

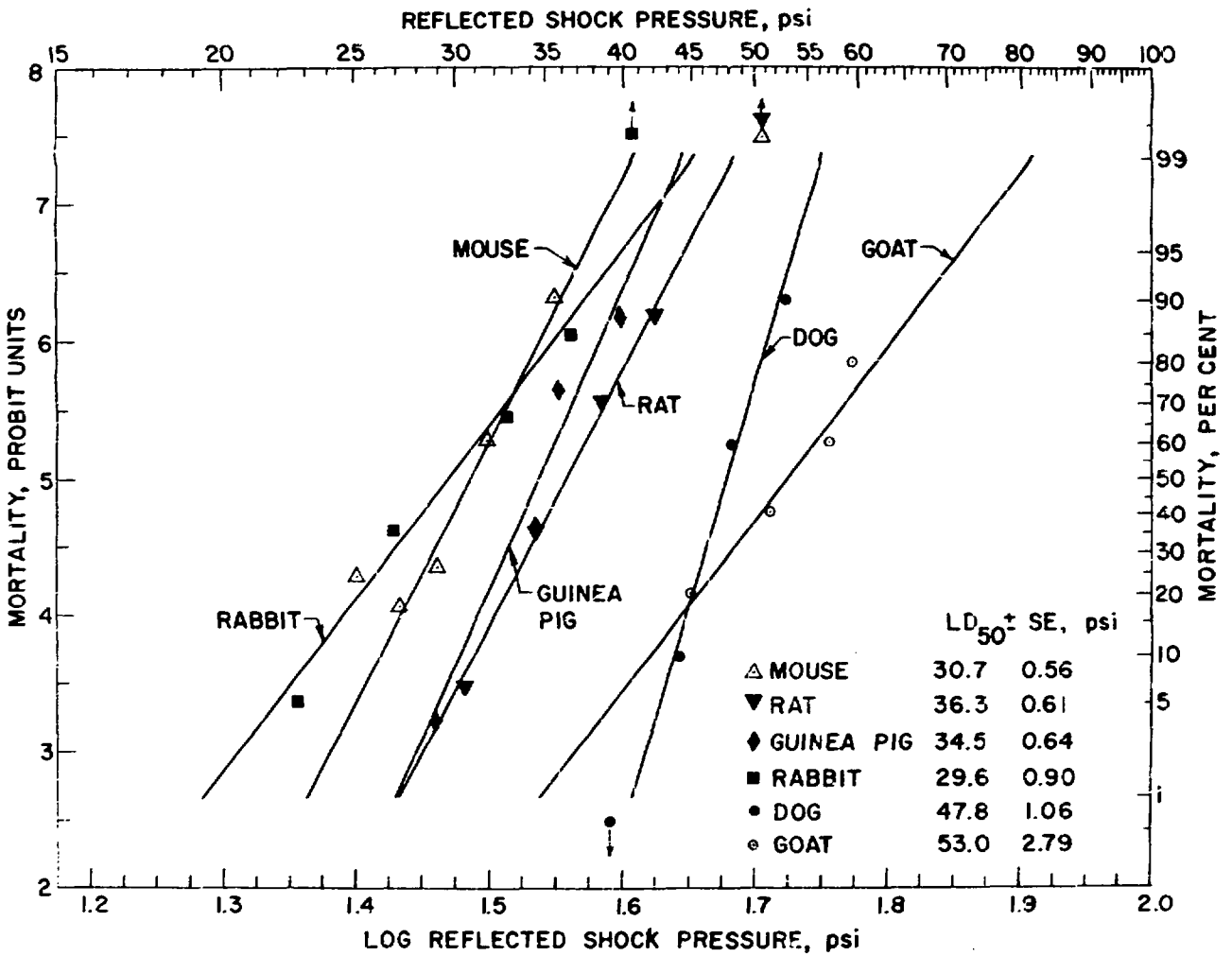

REFLECTED SHOCK PRESSURE, psi
15
20
25
30
35
40
45
50
55
60
70
80
90
100
MORTALITY, PROBIT UNITS
8
7
6
5
4
3
2
MORTALITY, PER CENT
99
95
90
80
70
60
50
40
30
20
10
5
1
MOUSE
GOAT
DOG
RAT
GUINEA PIG
RABBIT
LD50 ± SE, psi
△ MOUSE 30.7 0.56
▼ RAT 36.3 0.61
♦ GUINEA PIG 34.5 0.64
■ RABBIT 29.6 0.90
● DOG 47.8 1.06
⊙ GOAT 53.0 2.79
1.2
1.3
1.4
1.5
1.6
1.7
1.8
1.9
2.0
LOG REFLECTED SHOCK PRESSURE, psi

Fig. 23

RELATION BETWEEN BODY WEIGHT AND FAST-RISING OVERPRESSURES OF 400 MILLISECONDS DURATION NEEDED TO PRODUCE 50 PERCENT MORTALITY

REGRESSION EQUATION

$\text{Log } (LD_{50}) = 1.3673 + 0.06939 \log (BW)$

Where LD_{50} = Pressure required for 50% mortality, psi

BW = Average body weight of the group, grams

Standard Error of Estimate: 0.0602 log units (13.9 %)

Animals exposed side-on against the plate closing the end of a shock tube

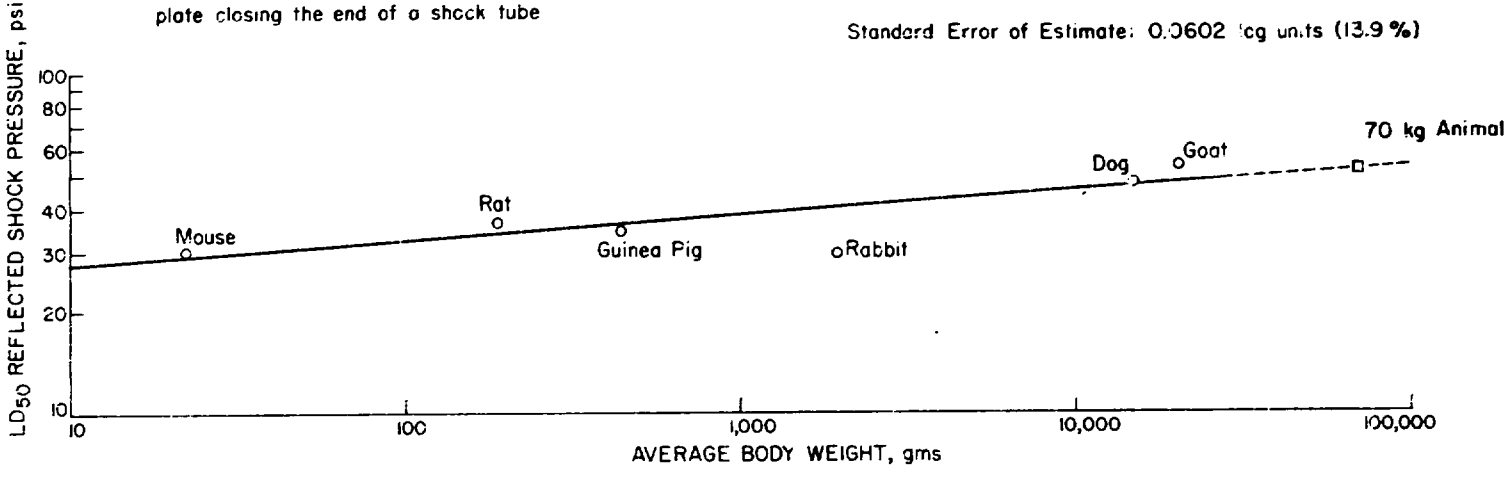


Fig. 24

Extrapolation of data to an animal of 70-kilogram weight yields a predicted P_{50} of 50.5 psi. For man today, the best estimate available for "long"-duration, "fast"-rising, single-pulse overpressures is 45-55 for the P_{50} and 35-45 and 55-65 psi for the P_1 and P_{99} range, respectively[4].

The threshold for lung hemorrhage is near 15 psi or about 6-7 psi incident in a geometry where maximum reflection can occur[2].

Similar studies were accomplished using different arrangements of the 40-inch tube and a specially designed 24-inch tube to obtain "fast"-rising, single-pulse overpressures enduring for 6-8 seconds in one study[25] and 3-4 milliseconds in the other[18]. Each series involved four species of animals, totaling 455 for the 6-8 seconds work, and 661 for the 3-4 milliseconds experiments.

In addition, tentative data are at hand for several hundred animals exposed to HE charges varying in weight from 4 ounces to 66 pounds[18,29]. For the latter experiments, animals were located on an instrumented concrete pad shown in Figure 25 directly below the charge. As in the shock tube, therefore, the animals were subjected first to the incident overpressure and, almost instantaneously, to the reflected pulse.

The results of the Albuquerque experiments using single-pulse, "fast"-rising overpressures ranging in duration from less than a millisecond to 6-8 seconds are shown as solid points in Figure 26 along with other HE data from the literature[18]. With exceptions that I will mention, all points represent the overpressure lethal to 50 per cent of animals and the figure relates this overpressure with duration of the pulse.

The German data of Desaga[9] for the P_{100} for dogs are shown as open semicircles, and that for the heifer as a plus.

This figure contains much information of worth and deserves comment as follows:

-21a-

Fig. 25

Lethality Curves for "Larger" and "Smaller" Animals

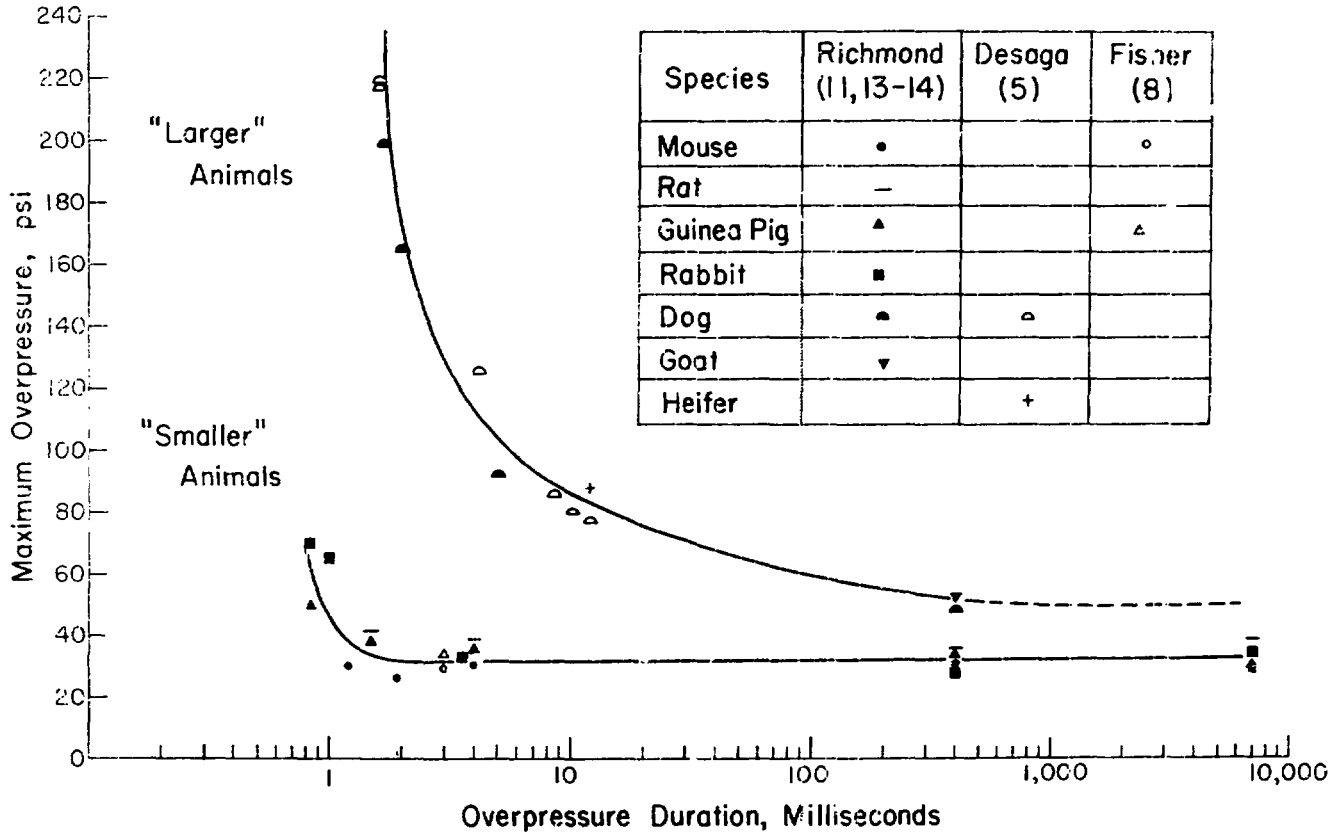


Fig. 26

a. Note that this data for the smaller and larger animals are separately grouped.

b. There is a critical pressure duration for each group less than which the killing pressure rises sharply and longer than which only the overpressure is definitive for lethality.

c. The critical duration bears a relationship to animal size. This duration is like a fraction to very few milliseconds for the smaller species and several to many tens of milliseconds for the larger animals.

d. Fourth, by way of comment, the P_{50} figures of Richmond[29] for dogs are consistently slightly less than those of Desaga which represent the P_{100} pressures also for dogs[9]. Since Desaga's experiments were done with animals and charge both on the ground, the animals were actually exposed to incident rather than reflected pressures. The Richmond and Desaga results are quite consistent. This means that the animal does not care whether the maximal overpressure is an incident pressure or an incident plus a reflected pressure providing the incident and reflected pulses are applied almost instantaneously.

e. Fifth, there is a spectrum of possibilities for extrapolation of animal data to mammals of man's weight. These depend upon the pressure-duration relationship we are now considering. This, in a way, is fortunate because it is consistent with the British contention[20] that man might tolerate up to 350 to 450 psi for the P_{50} for pulses of 1-3 milliseconds duration and the German results[9] indicating 235 psi as the more applicable figure also for small high explosive charges.

f. Sixth and last, let it be clear that for nuclear-produced overpressures, except for those from very small sub-kiloton yields, data referable to "long"-duration overpressures are those which apply to man[6].

These remarks apply only to "fast"-rising overpressures and unfortunately is not the entire primary blast story. Even the

well-integrated data we have been discussing need to be viewed with care as will be noted in more detail later. This is because conditions at the time of exposure may alter tolerance in at least two ways that are now known; namely, (1) if a stepwise increase in overpressure occurs, or (2) if the average rate of pressure rise is otherwise degraded. Too, it needs be pointed out that tolerance for the very young and very old has not yet been studied.

2. Step-loading

Because of the 1953 field observations wherein a stepwise increase in overpressure occurred, guinea pigs were exposed at various distances from the end-plate of a closed shock tube and the reflected pressure associated with the P_{50} was determined. Table 3 shows the data[3, 25] and the progressive increase in the P_{50} as distance from the end-plate increased is apparent; i. e., from about 37 psi against the end-plate to 41 at 1 inch, to 48 at 2 inches, to 56 at 3 inches, and to between 57 and 59 at 6 to 12 inches.

This increase in tolerance by over 50 per cent was associated with a quantitative variation among three variables; namely, an increase in magnitude of the incident pressure, the reflected pressure, and the time between arrival of the incident and reflected pulses.

To reduce the variables to 1 and to extend the work to other species, experiments were performed using incident and reflected overpressures of about 18 and 52 psi, respectively, a "load" which when applied "simultaneously" to animals exposed against the end-plate was 100 per cent fatal to mice, rats, guinea pigs and rabbits. Under these circumstances, only the time between the incident and the subsequent increase in pressure due to reflection was the variable.

Figure 27 shows the results[3, 25]. Outstanding is the fantastic ability of the animals to detect time differences. Mortality for the mouse, for instance, dropped from 100 per cent to 63 per cent at 1/2-inch association with a time interval of about 50 microseconds. At 1 inch, equivalent to 100 microseconds, mortality was 29 per cent and

TABLE 3

Mortality Data for Guinea Pigs for "Fast"-rising, "Long"-duration Shock Tube-produced Overpressures when the Incident and Reflected Overpressures are Applied in Two Steps (13, 14, 32)

Distance from end-plate in.	Number of animals	Overpressures associated with 50 per cent mortality in psi		Time between application of incident & reflected pressures msec
		incident	reflected	
0	140	12.1	36.7 ± 0.7*	0
1	75	13.4	40.8 ± 2.1	0.10**
2	78	15.6	48.3 ± 1.3	0.20
3	87	16.9	52.8 ± 1.9	0.30
6	99	18.7	58.6 ± 1.6	0.63
12	109	18.2	57.1 ± 1.1	1.36

*All plus or minus figures refer to the standard error of the mean
**Estimated

MORTALITY CHANGE WITH EXPOSURE

Distance from end-plate in.	Number Animals – Mortality in Per Cent							
	Mice		Rats		Guinea pigs		Rabbits	
	No.	%	No.	%	No.	%	No.	%
0	20	100	15	100	20	100	12	100
1/2	24	63	10	100				
1	24	29	15	80	15	100	4	100
2	15	0	15	13	18	72	8	88
3	15	7	15	0	30	37	8	63
6			15	0	21	24	6	17
12			5	0	15	25	2	0

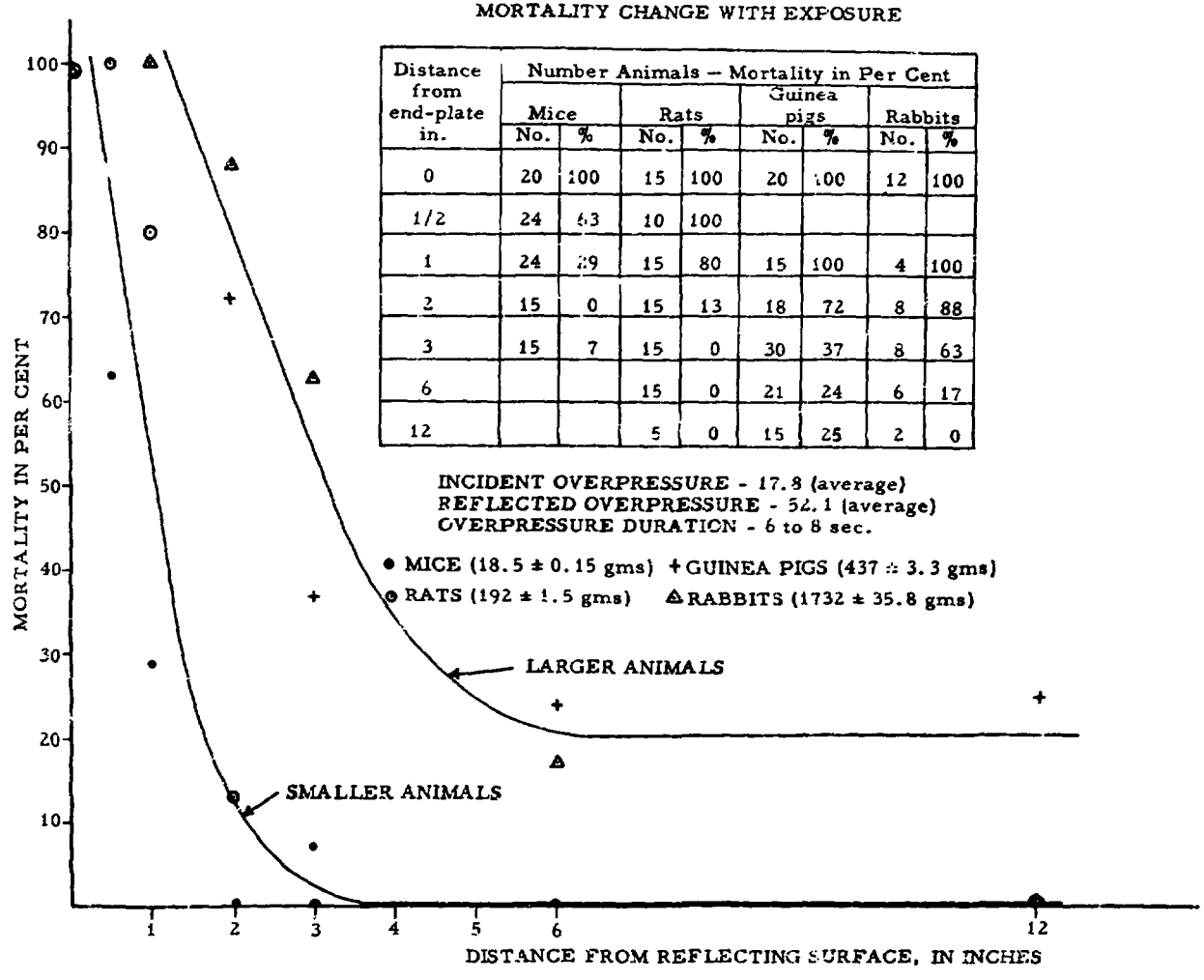


Fig. 27

zero at 2 inches, where a 200-microsecond interval separated the two steps comprising the pressure pulse.

Each of the other species exhibited similar behavior, though the larger the animal, the more slowly mortality decreased with increasing distance from the end-plate. Only the guinea pigs failed to drop to zero mortality.

Another way to express this rather startling result is to say that for very short separations in time — like 200-400 microseconds — between arrival of the incident of reflected pulses, the animal "appreciates" them as 1 pulse. This is so because mortality is higher than it would be from either of the pulses applied alone. For periods longer than this, the animal makes an adaptation such that application of the first pulse protects him from the second. This is so because mortality is less than it would be were either applied separately.

Though this result is partly understood and cannot be discussed further here, it is none the less true that the step-load problem has not been studied in dogs or larger animals, and what may be the implications for human tolerance only now can be summarized. For sure, it is an important problem for future research.

3. Slowly Rising Overpressures

Several years ago a few dogs were exposed to "slowly"-rising overpressures of 5 to 10 seconds duration, as shown in Figure 28[27]. The pressures rose to a maximum in 150, 90, 60, and 30 milliseconds, and though sinus hemorrhage and eardrum rupture occurred, gross damage to the lung was minimal and consisted of marginal bruising, apparently because the lung was caught between the upward-moving diaphragm and the inward-moving chest wall. Though overpressures

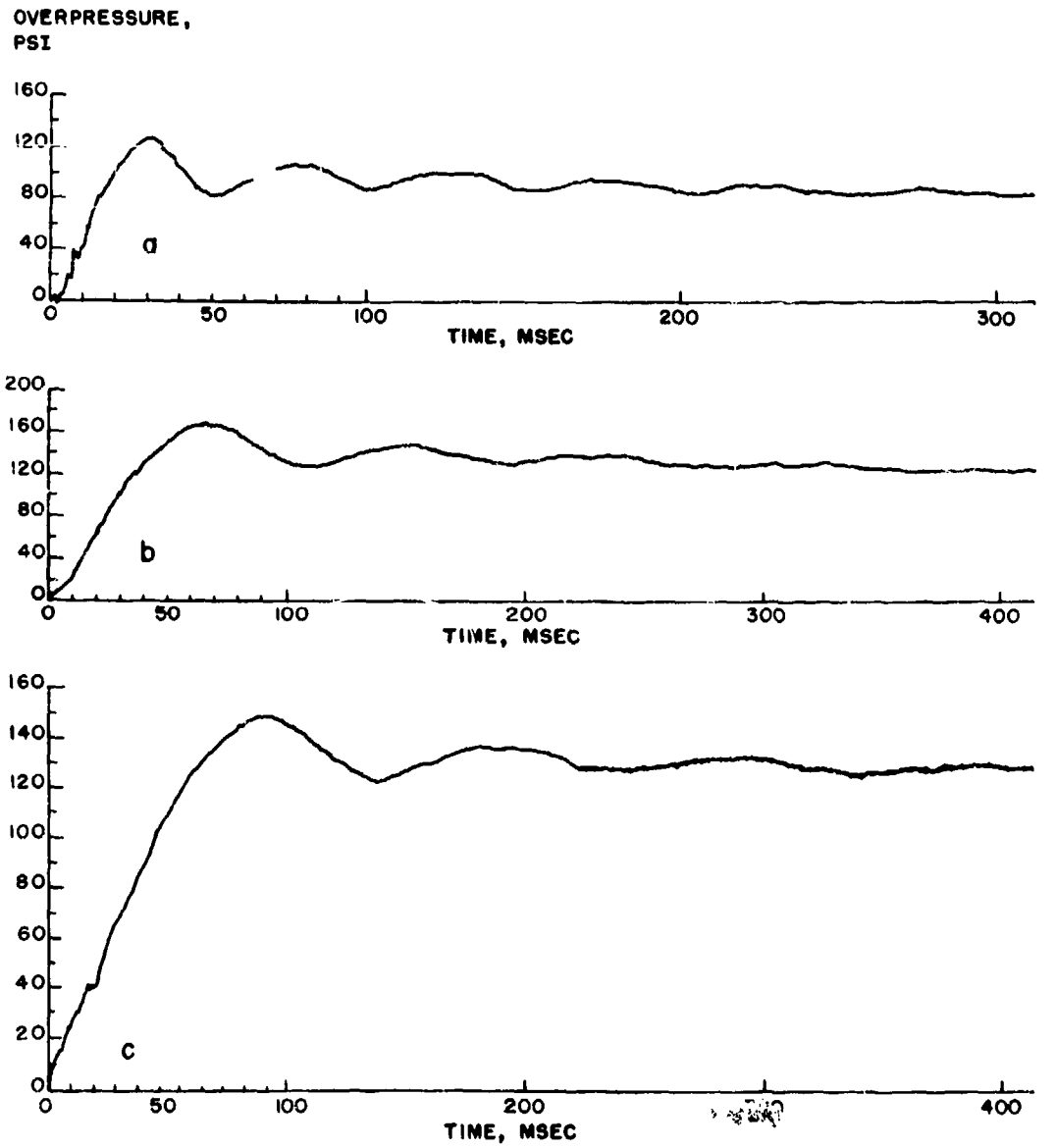

OVERPRESSURE,
PSI
160
120
80
40
0
a
0
50
100
200
300
TIME, MSEC
200
160
120
80
40
0
b
0
50
100
200
300
400
TIME, MSEC
160
140
120
100
80
60
40
20
0
c
0
50
100
200
400
TIME, MSEC

Fig. 28

ranged from 30 to 170 psi, no lethalities or even typical blast-like lung lesions were observed. The highest overpressures employed were between three- and fourfold known to be fatal for "fast"-rising overpressures even when the pulse duration is quite prolonged.

In a few experiments with times to maximum pressure of between 20 and 30 milliseconds and with overpressures in excess of 140 psi, Richmond[3, 29] observed "blow-out" fractures of the orbital walls in dogs. Figure 29 shows one specimen. The fractures are apparently into the nearby sphenoid and ethmoid sinuses and occur because the sinus ostia delay the arrival of pressure to the sinus-side of the orbital bones to counter the "fast"-arriving pulse transmitted through the fluid content of the orbit. A complication of this lesion can be intra-orbital hemorrhage with proptosis of the eyeball, a rare but startlingly wierd sign of blast exposure in man. Too, fracture lines may extend upwards into the cranial vault and open a route for infection of the meninges.

4. Pathophysiology

Before leaving the primary blast problem, there are a few other points of great interest; namely, the time of death in mortally-wounded animals and the nature of the pathologic damage produced.

a. Time of death - Figure 30 shows the per cent of 287 animals lethally wounded by blast plotted as a function of time[18]. These animals were among the 661 employed in the 3-4 millisecond study mentioned earlier. Note that mortality occurs quickly, being about 50 per cent complete in 5 minutes, near 70 per cent complete in 15 minutes, and close to 90 per cent complete in 30 minutes.

Fig. 29

Cumulative Percent of Mortally Wounded Animals Dying Over a Two Hour Period From Exposure to "Sharp"-Rising Overpressures of 3-4 Milliseconds Duration

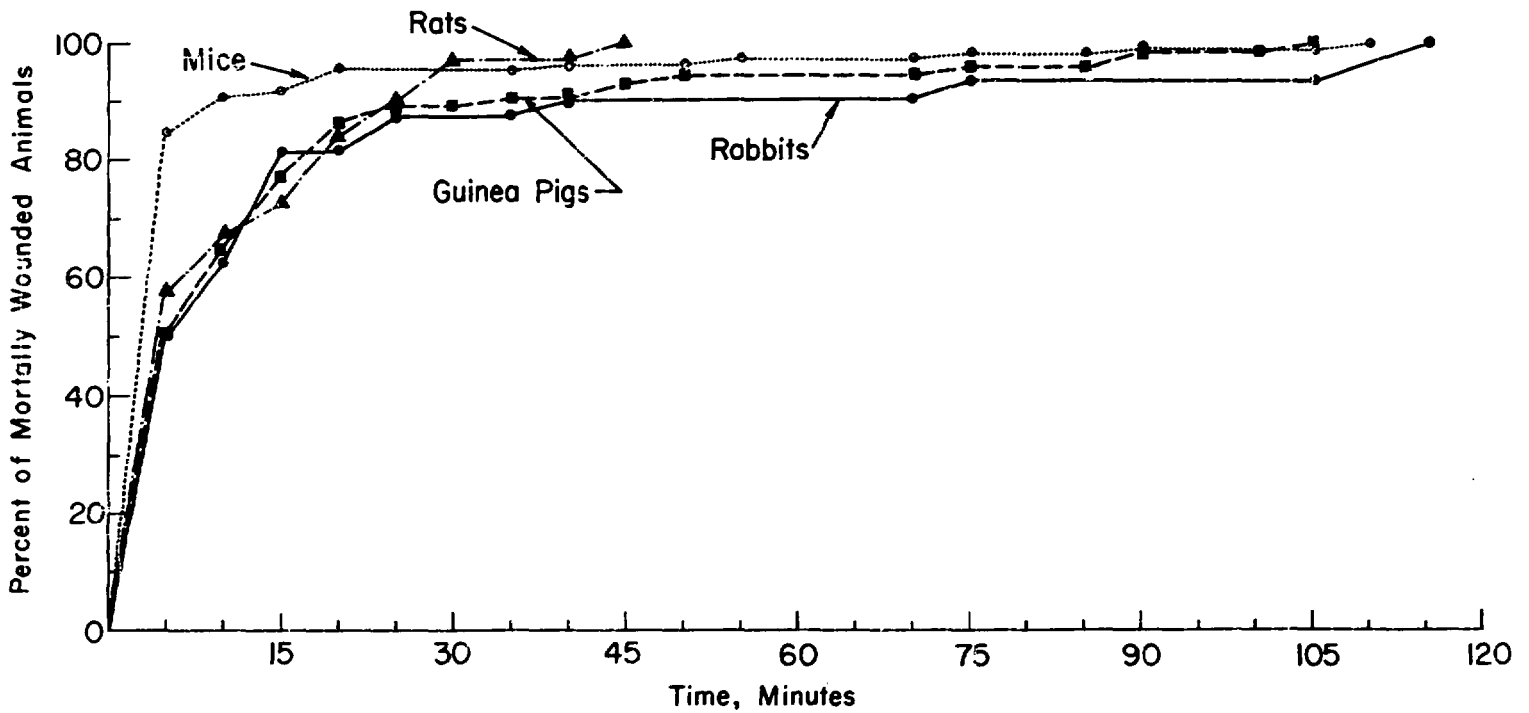


Fig. 30

Figure 31 shows a survival curve for a series of untreated and blasted guinea pigs followed for 30 days[29]. Out of 100, a total of about 30 and 45 died in 1 and 24 hours, respectively. An additional 10 animals succumbed over the following 15 days. There were no additional deaths after the 17th day.

It is clear that primary blast injury is very hazardous indeed and, therefore, represents a type of injury to be avoided at all costs.

b. Gross Pathology - It is definitely true, as implied earlier, that damage to the animal from "fast"-rising overpressures occurs at those areas in the body where the variations in tissue density are the greatest. The special target organs are those containing air; e. g., the paranasal sinuses, the ears, the GI tract, and particularly, the lungs. In addition, there are signs and symptoms which occur at a distance from these organs which are due to air emboli. Any organ in the body may be involved.

In air blast, rupture of the ear and bleeding from the sinuses is not an immediate threat to the organism. Rupture of the abdominal viscera is relatively rare, unless displacement and impact also occur. However, abdominal pathology is more prominent in underwater blast casualties. Bleeding into and edema of the lungs are common and very dangerous, but most hazardous of all, are ruptures of the walls between the alveoli and the pulmonary vein which allow air emboli to reach the heart and general circulation.

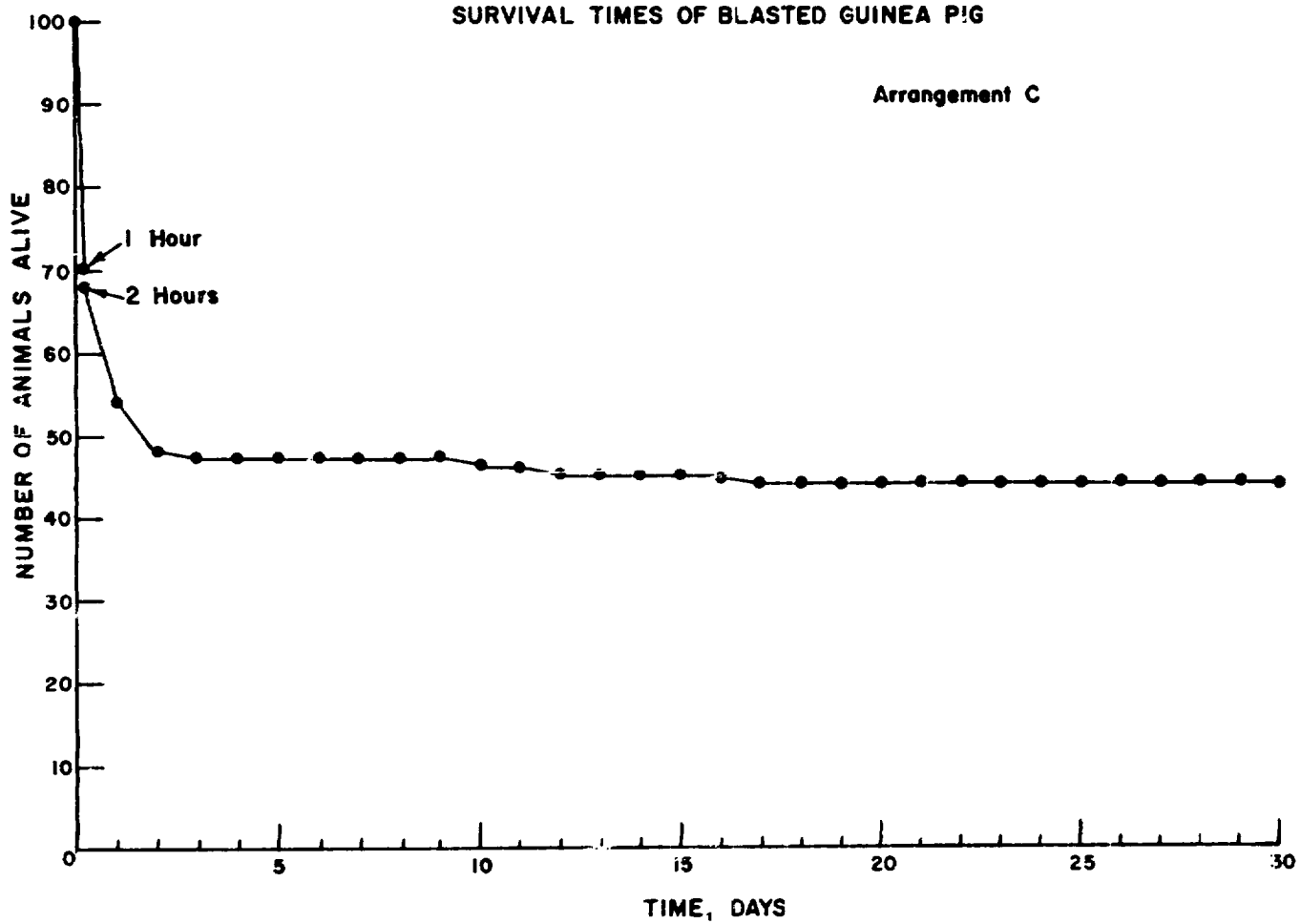

SURVIVAL TIMES OF BLASTED GUINEA PIG
Arrangement C
NUMBER OF ANIMALS ALIVE
100
90
80
70
60
50
40
30
20
10
0
1 Hour
2 Hours
5
10
15
20
25
30
TIME, DAYS

Fig. 31

Almost immediate death can occur from interruption of the circulation to the myocardium, or to critical portions of the central nervous system. Animals that manage to survive the first few minutes face the hazards of continuous bleeding and edema of the lung and the sequallae of heart damage from multiple small air emboli. Exercise after a significant blast exposure is very, very dangerous and is to be avoided if at all possible.

It will be of interest to present a few figures illustrating the statements just made.

Figure 32 shows the lungs from a normal, healthy dog.

Figure 33 illustrates the damage that occurs at density interfaces[3]. The centrally-located, dark area is the hemorrhagic image of the heart where it was in contact with the lung. The specimen is from a non-fatally blasted dog.

Figure 34 is a photograph of the lungs of a fatally-injured dog and shows very well the marked and widespread hemorrhage which occurs[3].

Figure 35, a picture of the lungs of a pig that succumbed to blast injury, shows the characteristic rib markings quite distinctly[7]. These, like the "heart image" noted previously, illustrate the location of pathology at the interfaces where tissue density variations are great.

In marked contrast are the lungs of animals exposed to "slowly"-rising overpressures[27], a photo of which is shown in Figure 36. Note the marginal areas of hemorrhage which are the only significant lesions grossly apparent.

Figure 37 shows the heart does not escape damage where it is in contact with the lung as evidenced by the bruised area just to the left of the central portion of the picture[3].

Most dramatic, however, are the large and numerous air emboli visualized in the coronary arteries as well demonstrated in

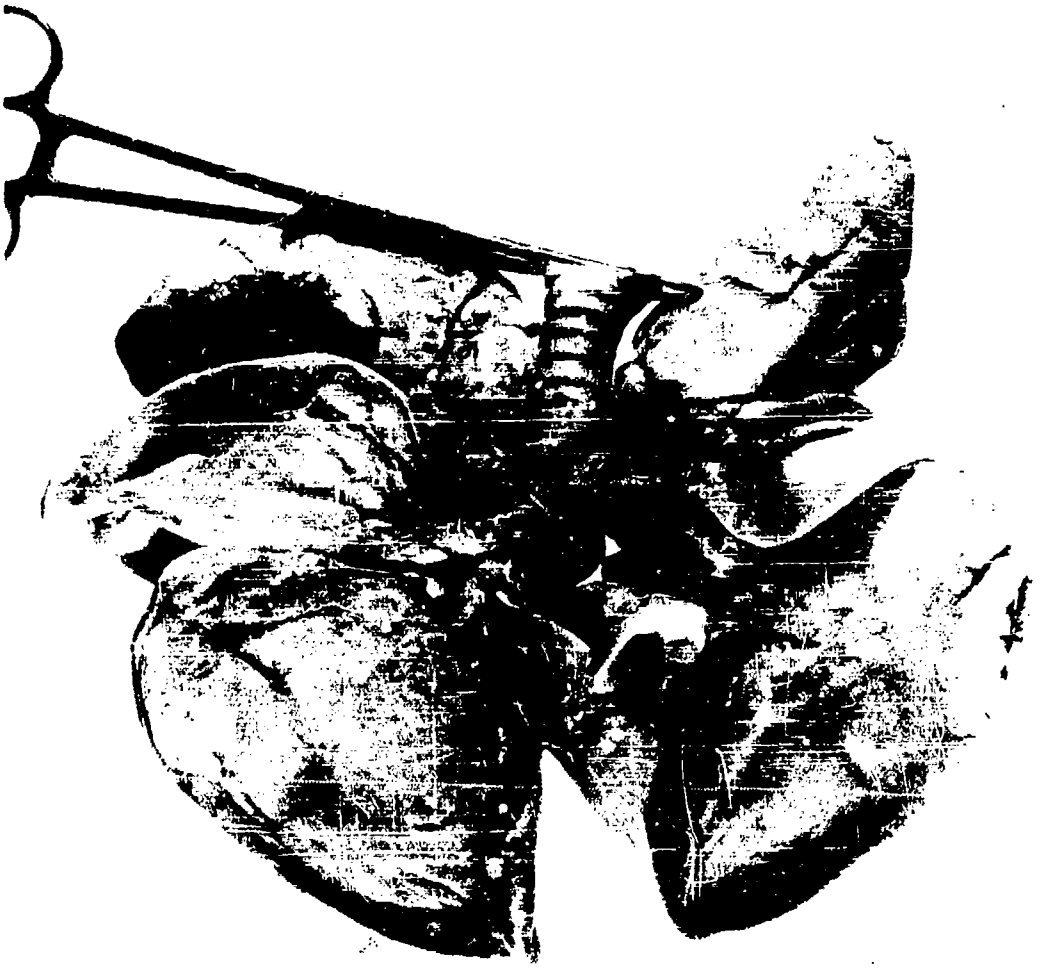
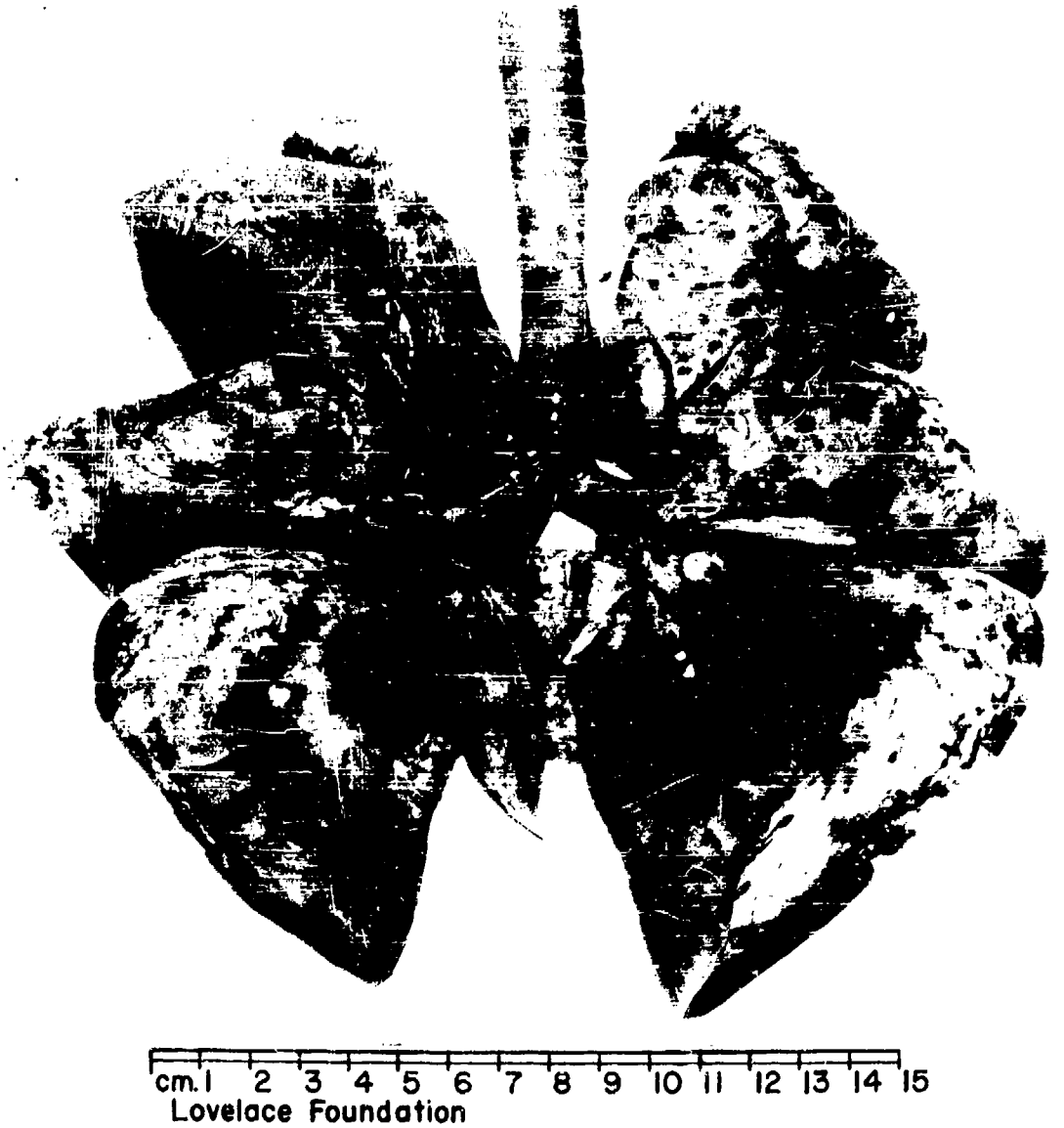


Fig. 32



T-41
4-24-58

Fig. 33

Fig. 34

PIG 7

Fig. 35

ANIMAL T/8 · 11-12-56
170 PSI · RISE TIME- 60 MSEC.
DURATION - 10 SEC.

Fig. 36

Fig. 37

Figure 38 which reproduces what can be seen frequently in the vessels of the canine heart[3]. These findings are common to all species that have been investigated and have been described in man. Air emboli are also frequently visualized in the superficial cerebral arteries after removal of the calvaria.

Figure 39 shows a microphotograph of the lungs of a blasted dog[30]. The alveoli are relatively free of hemorrhage, but the centrally-located pulmonary vein shows the characteristic separation of its wall structures. Note the delicate structure of the small vascular tributary where it lies in contact with the lace-like small air sacs, the alveoli.

Figure 40 shows a microsection of a hemorrhagic lesion in the lung of a fatally-injured dog for which I am indebted to Dr. Thomas L. Chiffelle, the Chief Pathologist of the Lovelace Foundation[30]. It is a classic and the only one of its kind I have ever seen. There is failure of the wall of the pulmonary vein, and escape of blood into the surrounding lung is clearly demonstrated. It is in such locales that air no doubt enters the blood stream to be carried to the left heart and hence to the peripheral circulation, a consequence of which can be almost immediate fatality.

So much for a brief and somewhat hurried look at the primary blast problem.

Let us now turn to the task of summarizing recent experiments first, in the secondary and then in the tertiary blast area, aimed at evaluating at least some of the serious consequences of impact.

B. Secondary Blast Effects

Biologically, injury from secondary missiles depends upon a host of variables, including the velocity of the missile and its angle at impact, its mass, density and shape, and the area or portion of the body traumatized either from penetrating or nonpenetrating debris. Since the biology of high-velocity ballistics has been much investigated and because very little was known about relatively low-velocity debris,

Fig. 38

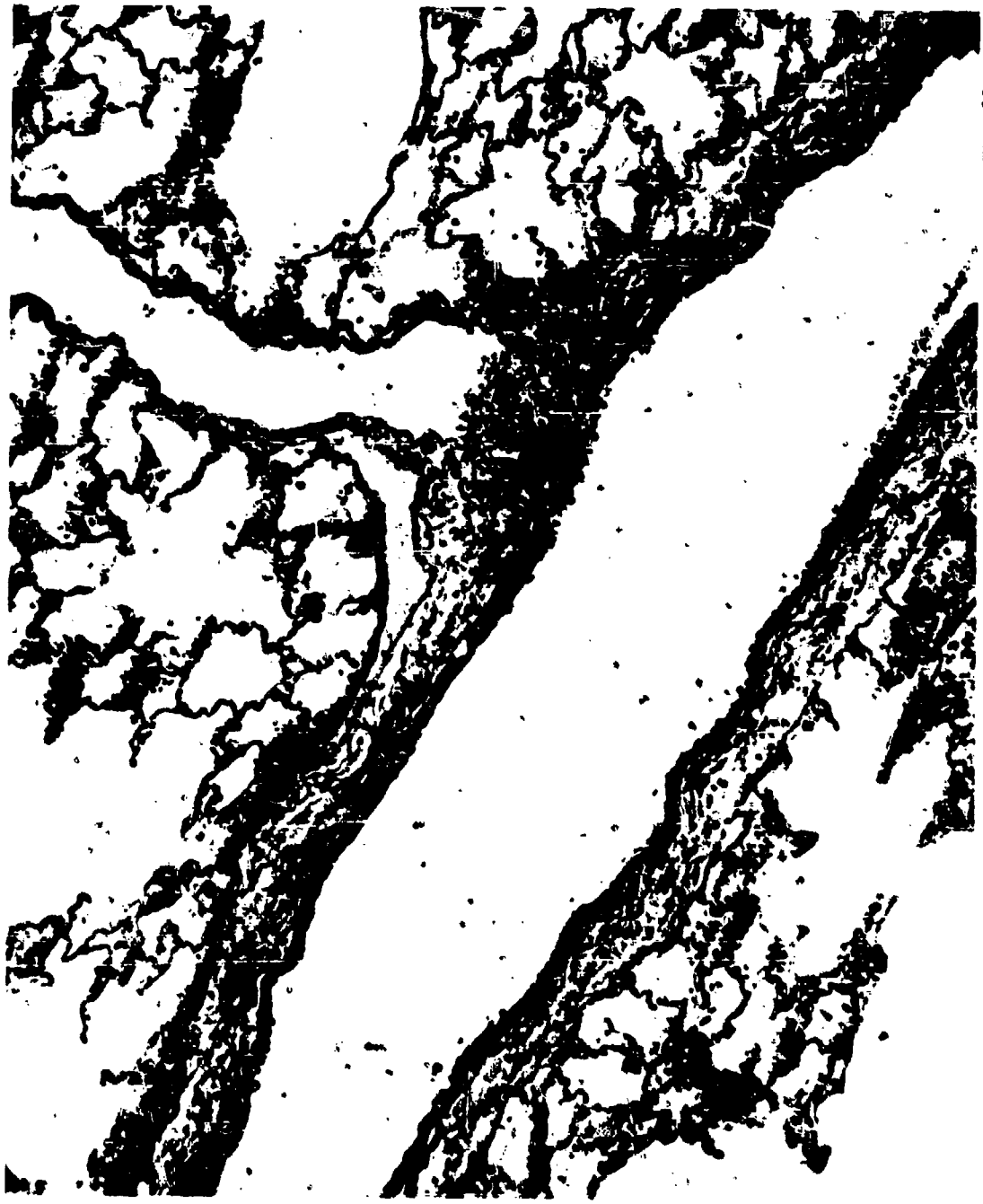


Fig. 39

Fig. 40

experiments were undertaken with glass-fragments ranging in weight from about 0.1 to 2 grams. Such missiles were energized by an air gun, and, as a function of impact velocity and weight, the probabilities of a fragment piercing the abdominal wall and entering the peritoneal cavity were determined. Dogs were employed and the average thickness of skin and soft tissue penetrated was about 1 centimeter.

The results are presented in Figure 41 which shows probabilities of penetration from zero to 100 per cent as curves relating missile impact velocity to missile mass[31]. Note that the chances of penetration are very small at velocities ranging from about 100 to 300 feet per second and quite high from about 300 to 1000 feet per second depending upon missile mass. Such data were used to give a crude evaluation of the 1955 field missile studies[32], and field experiments with animals in 1957[33] proved fairly consistent with the laboratory work.

From such studies and an analysis of the Japanese data, one can tentatively propose criteria for glass missiles. These are that:

1. Skin lacerations may be anticipated at missile velocities in the order of 50 feet per second and,

2. Serious wounds involving penetration of serous cavities may be predicted at velocities of about 100 feet per second in a few cases and in most cases above 400 feet per second.

It is significant that these criteria can now be related to nuclear explosion of various yields as noted earlier.

Let it be quite clear that such criteria are very crude and only tentative, that no studies with glass in relation to the eyes have been made (though Stewart in the Ballistic Laboratory at the Chemical Corps[34] has done so with steel cubes and spheres employing the rabbit's eye as a target), and that the protective effects of clothing for low-velocity debris is not well understood.

With regard to nonpenetrating missiles, the head appears to be the critical organ with the possible exception of impact over the liver and spleen. For blunt objects of about 10-pounds weight (near that of

Probability of penetration of glass fragments into the abdomen of a dog as a function of missile mass and impact velocity.

Equation: $\log v = 2.5172 - \log(\log m + 2.3054) + 0.4842 P$

where v = impact velocity in ft/sec
m = mass of glass fragments in gms
P = probability of penetration

Standard Error of Estimate: 0.0745

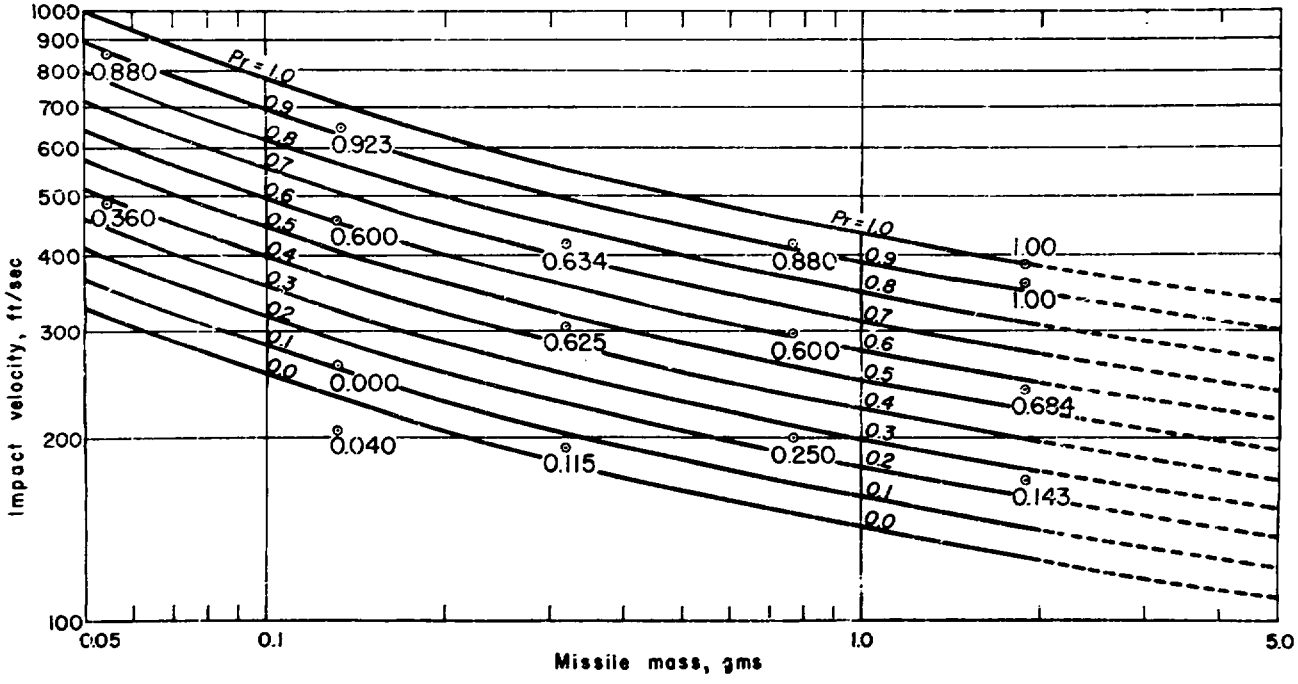


Fig. 41

the adult human head), velocities of 10 feet per second seem to be relatively safe; skull fracture is likely at 13-14 feet per second and will be the usual thing at impacts above 20-22 feet per second[35].

Though these data are fragmentary and more is known than summarized here, enough has been said to give a general feel for the problem. Too, those interested can consult CEX-58.8[4] for additional information and references.

Now let us turn to the tertiary blast area.

C. Tertiary Blast Effects

Though an animal or man bodily hurled through the air may be damaged because of differential displacement of different portions of the body during the general process of acceleration, it is known that the decelerative experience of stopping can be far more dangerous. It is clear that the character of the decelerating surface, the angle and area of the body involved at impact, the impact velocity, and the decelerating time and distance are each critical factors. Most hazardous of all (with certain rare exceptions) is, in all probability, uncoordinated impact against a very hard surface.

Because little or no quantitative data were at hand referable to impact under circumstances where only the animal's own tissues were "active" in absorbing energy, an interspecies study was initiated several years ago and only finished and published recently[36].

A total of 455 animals were involved, among which were 113, 178, 111 and 53 mice, rats, guinea pigs and rabbits, respectively. Each was subjected to ventral impact by a drop onto a flat, concrete surface. The height of drop was varied to obtain different impact velocities.

Mortality curves relating per cent lethality to impact velocity were determined and the velocities associated with 50 per cent lethality in 24 hours were calculated. The figures were about 39, 44, 31 and 32 feet per second for the mouse, rat, guinea pig and rabbit, respectively.

A plot of these data as a function of average body weight for each species is shown in Figure 42, along with the regression equation obtained by the least squares technique[36]. The standard error of the estimate was 0.42 log units or 9.7 per cent.

Extrapolation of the results to the 70-kilogram animal predicted a 50 per cent lethal velocity of 26 feet per second or 18 miles per hour.

This turned out to be an encouraging result in view of a number of data in the literature referable to humans. First, for example, fatalities of 40 per cent have been reported for urban automobile accidents associated with estimated vehicular speeds of 20 or less miles per hour (28 feet per second). Also, a 70-per-cent-mortality figure has been associated with speeds of 30 or less miles per hour (41 feet per second)[4,37]

Second, the impact velocities for fracture of the human skull obtained by Gurdjian[2,35] in experiments wherein "fresh" human heads were dropped onto a solid surface ranged from 13-14 feet per second (9-10 miles per hour) to 21-23 feet per second (15-16 miles per hour) is shown by Table 4[2].

Third, fractures of the heel bone, feet and lower extremities have been reported at impact velocities of 11-16 feet per second for hard surfaces with the knees locked[4,36,38].

Fourth, Swearingen et al.[39] have recently published a paper noting that about 10 feet per second was the voluntary tolerance of human volunteers subjected to impact in both the sitting and standing positions.

Thus, it is possible to regard the figure of 10 feet per second as "safe" and to believe tentatively at least, that human injury may occur at velocities much above this; that mortality may, on the average, become significantly frequent for "uncoordinated" impact at velocities between 15 and 20 feet per second, fairly common between 20 and 30 feet per second, and near 100 per cent fatal between 30 and 40 feet per second, providing impact occurs with a hard surface where stopping distance is quite small and the stopping time is almost instantaneous.

IMPACT VELOCITY ASSOCIATED WITH 50 PERCENT MORTALITY
AS A FUNCTION OF AVERAGE BODY WEIGHT

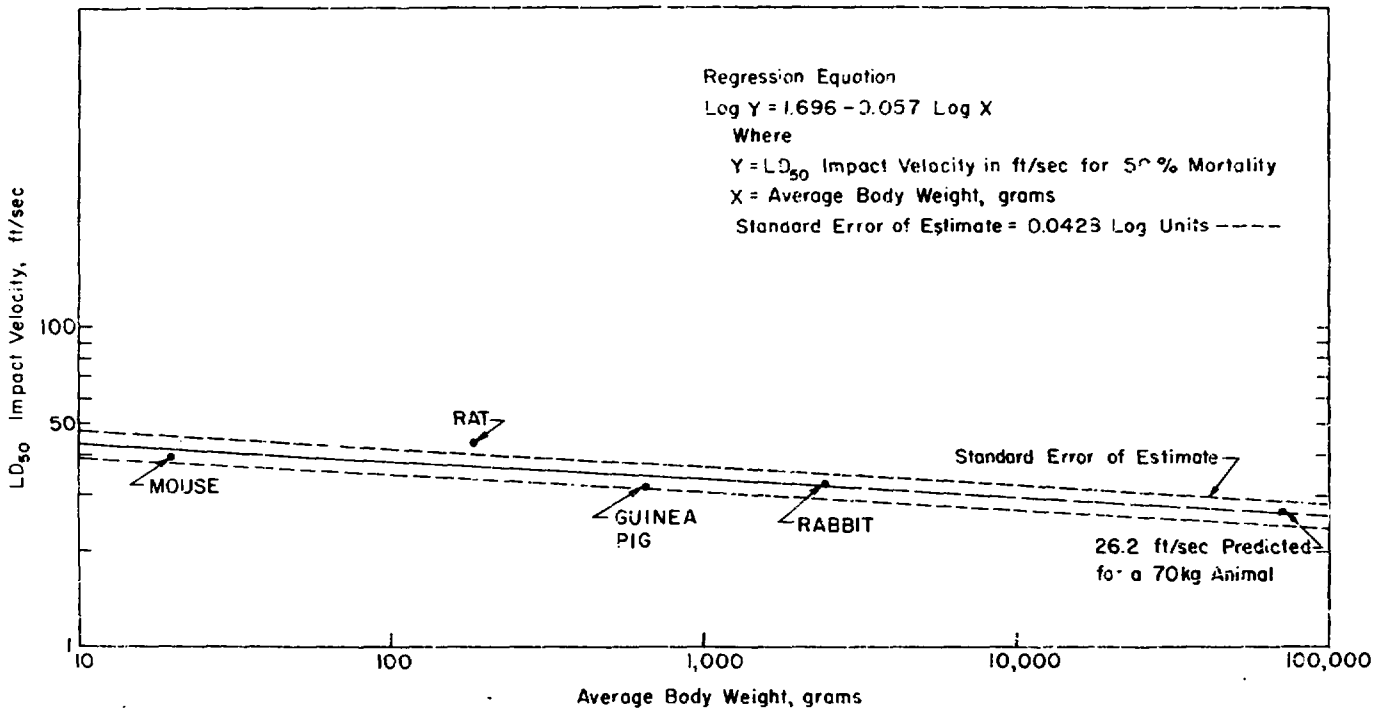


Fig. 42

Table 4

The Ranges of Impact Velocities Associated with Experimental Fracture of the Human Skull

Range impact velocities ft/sec	Approx. velocity in mph	Approx. height of fall in.	Number of subjects	Fractures in per cent
13.5-14.9	9.5	37	9	19
15-16.9	10.9	48	10	22
17-18.9	12.2	61	12	26
19-20.9	13.6	75	11	24
21-22.9	15.0	91	4	9
Total			46	100

Minimum velocity with fracture - 13.5 ft/sec (9.2 mph)
Maximum velocity with fracture - 22.8 ft/sec (15.5 mph)
Maximum velocity without fracture - unstated.

While the cause of impact death in experimental animals is under study using the gross pathological findings noted in the study just described, it is not possible now to specify the lesions whose incidence parallel the incidence of mortality and bear the appropriate relation to impact velocity.

One significant finding is clear, however, and this concerns the time of death of 200 untreated animals mortally wounded by impact.

Figure 43 gives the data[36]. Apparently, impact lethality – like that associated with primary blast injury – is characteristically rapid, being near 50 per cent complete in 5 to 30 minutes and about 90 per cent complete in 1 to 2 hours.

Whatever the etiology involved, violent impact posed a serious challenge to the four species of animals studied and no doubt – as the automobile accident figures annually show – also does so for man. Without question, such data focus attention on the need for medical care that is appropriate both in kind and in time.

In the context of a nuclear war, impact injury, like is the case with primary and secondary blast effects, is best avoided through making every effort to arrange and control the conditions of exposure.

V. Survival Data from Hiroshima

It is instructive, informative, and both optimistic and depressing to consider selected survival data applicable to Hiroshima which are available in the reports of the Joint Commission[40] and the excellent volume authored by Oughterson and Warren[41]. Your attention is directed to Figure 44 prepared using data from the sources mentioned, which with the central dotted curve, shows the over-all per cent survival as a function of range from the hypocenter along with survival curves under different conditions of exposure. The latter includes three groups of individuals; namely,

1. On the far right, school personnel in working parties who were mostly in the open at the time of the detonation;

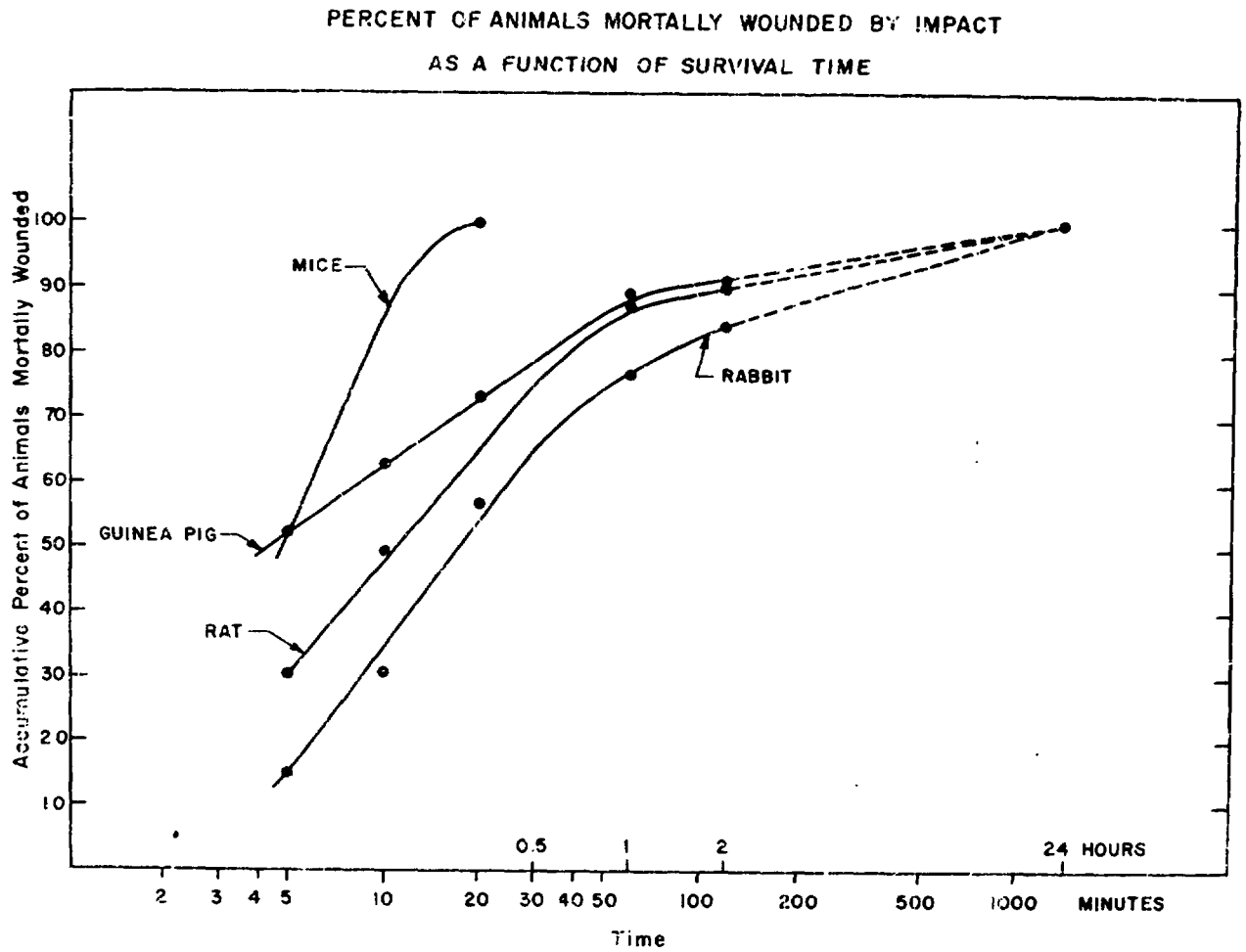

PERCENT OF ANIMALS MORTALLY WOUNDED BY IMPACT
AS A FUNCTION OF SURVIVAL TIME
Accumulative Percent of Animals Mortally Wounded
100
90
80
70
60
50
40
30
20
10
MICE
GUINEA PIG
RAT
RABBIT
0.5
1
2
24 HOURS
2
3
4
5
10
20
30
40
50
100
200
500
1000
MINUTES
Time

Fig. 43

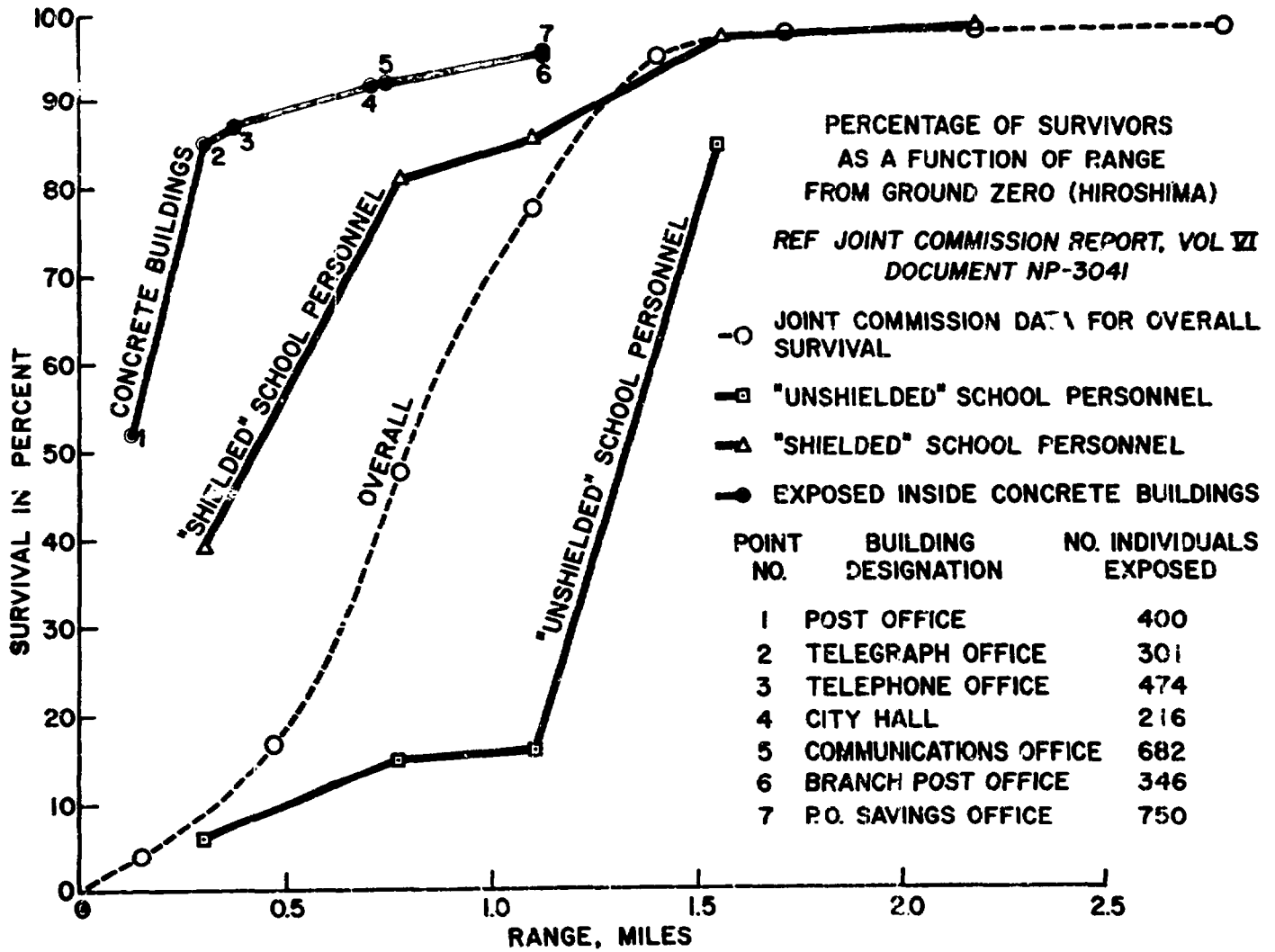

PERCENTAGE OF SURVIVORS
AS A FUNCTION OF RANGE
FROM GROUND ZERO (HIROSHIMA)
REF JOINT COMMISSION REPORT, VOL VI
DOCUMENT NP-3041
JOINT COMMISSION DATA FOR OVERALL SURVIVAL
"UNSHIELDED" SCHOOL PERSONNEL
"SHIELDED" SCHOOL PERSONNEL
EXPOSED INSIDE CONCRETE BUILDINGS
POINT NO. BUILDING DESIGNATION NO. INDIVIDUALS EXPOSED
1 POST OFFICE 400
2 TELEGRAPH OFFICE 301
3 TELEPHONE OFFICE 474
4 CITY HALL 216
5 COMMUNICATIONS OFFICE 682
6 BRANCH POST OFFICE 346
7 P.O. SAVINGS OFFICE 750
CONCRETE BUILDINGS
"SHIELDED" SCHOOL PERSONNEL
OVERALL
"UNSHIELDED" SCHOOL PERSONNEL
SURVIVAL IN PERCENT
RANGE, MILES
0
10
20
30
40
50
60
70
80
90
100
0.5
1.0
1.5
2.0
2.5

Fig. 44

2. The curve in the left central area marked by triangles applies to school personnel mostly inside schools when the explosion occurred; and

3. On the far left is the survival curve for over 3000 individuals located inside concrete buildings at burst time. Survival here applies to individuals known to be alive 20 days postshot.

There are a number of simple lessons portrayed by these survival curves which actually relate human experience with a nuclear detonation. Let us consider some of the more important.

1. First, the 50 per cent survival ranges for the four curves from your right to left of 1.3, 0.8, 0.45 and 0.12 miles forcefully emphasizes the importance of the conditions of exposure.

2. The area of complete destruction at Hiroshima has been described as covering a circle of about 1.2-mile radius (4 square miles), a range at which 4-5 psi existed. At this range there was an over-all survival of near 90 per cent. It is apparent, therefore, that one must not confuse the area of complete destruction of houses (a physical concept) with "complete destruction" of people. Even in to near 0.2 mile, there was 5 per cent over-all survival. By way of emphasis, let it be clear that there was a marked difference between the ranges for physical and biological destruction at Hiroshima. The gloomy habit of confusing the two concepts is, I am afraid, as prevalent as it is unrealistic and, indeed, untrue.

3. The great good fortune of just being indoors and shielded against the most far-reaching effect, direct thermal radiation, is illustrated by the survival range of 0.45 mile for school personnel mostly inside compared with 1.3 miles for those mostly outside. This proved so even though the fact of being inside involved exposure to falling and flying debris, greater displacement potential and higher pressure reflections. Apparently, the latter hazards are relatively less than the dangers from direct thermal radiation.

4. The marked value of simply being inside concrete buildings is illustrated by the 50 per cent survival range of 0.12 mile. To me,

this is a remarkable piece of information. There were 400 individuals inside the forward building, the Post Office. Two hundred became casualties almost immediately, no doubt mostly because of primary, secondary and tertiary blast effects. The remaining 200 were alive 20 days later[40, 41]. Though many, no doubt, subsequently succumbed because of exposure to ionizing radiation, the effective shielding against direct thermal radiation, blast pressures, winds and debris is quite clear. There was nothing special about the building except it was built to seismic codes.

5. The illustrated progressive decrease of the range for 50 per cent survival from 1.3 to 0.12 miles — about a factor of 10 — as it varied with conditions of exposure occurred by accident in Hiroshima. This fact is worth emphasizing and contemplating for at least three reasons; namely,

a. The potential utility of warning is amply demonstrated. Think of the differences in casualties which might have occurred in Hiroshima had the population just been mostly indoors.

b. The potential value of planning the conditions of exposure for shielding against immediate effects is made crystal clear. This statement is meant to apply from simple measures, such as being inside concrete buildings, provision of "hard" areas in houses, preparation of backyard and basement "cyclone" shelters, to design and construction of more complex underground shelters built to withstand 100-200 or more psi.

c. The great difference there can be between weapon-induced environmental variations outside and inside structures is sharply highlighted. It is indeed unfortunate that more is not known about "geometric"- compared with "free-field"-scaling, though admittedly if one grants that protection is best achieved by closed underground shelters, then the problem involves mostly that of shielding against ionizing radiation, both initial and residual.

Be this as it may, it will be instructive to spend a few moments considering the "free-field" blast parameters that may be scaled

for a 20-kiloton yield burst at 1850 feet using data from The Effects of Nuclear Weapons[15]. Table 5 segregates such information for the 50 per cent survival ranges applying to the curves we have been considering. Let it be clear that I am not, at the moment, interested in exact numbers for each effect, but rather the relative relationships.

Consider first the incident overpressures of 3-4 psi associated with 50 per cent survival for individuals outdoors, 7-8 psi for the over-all average, 15-20 for persons inside school houses, and 30-40 for concrete buildings. Even if the figures were too high by 20 to 50 per cent, these data are quite valid and to me as instructive as they are startling.

Now note all the "free-field" parameters at the 1.3-mile range in terms of biological significance; e.g.:

1. 3.6 - 7.9 incident and reflected psi – this might rupture a few eardrums; but very few would have lung injuries from pressure variations.

2. Winds of 170 miles per hour, along with the pressure-duration-yield parameters, allow one to predict a displacement velocity – at 10 feet of travel – of 9 feet per second for man and 115 feet per second for glass. The former represents no particular hazard, but the glass missile velocities do. Indeed, in Hiroshima glass laceration occurred out to a little over 2 miles.

3. The thermal flux of 9 cal/cm^2 is well above the thermal fluxes of 2.5, 5 and 7 cal/cm^2 required for first, second, and third degree burns, respectively, for a 20-kiloton yield[6].

4. The initial ionizing radiation flux of 15 rems in an emergency represents no hazard.

In contrast, estimated "free-field" effects at the 0.12-mile range look very formidable, e.g.:

1. The incident and reflected overpressure of 37 and 130 psi with a pulse duration in excess of 100 milliseconds are sufficient to cause

TABLE 5

Comparative "Free"-field Effects Parameters at 50 Per Cent Survival Ranges for Over-all and Other Conditions of Exposure at Hiroshima*

Items of Interest**	Conditions of Exposure			
	Concrete buildings	Inside schools	Mixed (average)	Outside schools
Range for 50% survival - mi	0.12	0.45	0.8	1.3
Estimated "free"-field effects at range for 50% survival				
Max side-on overpressure - psi	37	20	7.9	3.6
Max reflected overpressure - psi	130	59	19	7.9
Duration of overpressure - sec	<0.15	0.21	0.26	0.35
Max wind - mph	780	500	240	170
Thermal radiation - cal/cm^2	140	58	24	9
Initial ionizing radiation - rems	59,000	5800	480	15
Max displacement velocity at 10 ft of travel - ft/sec				
10-gm glass fragment	>400	>400	355	115
165-lb man	> 40	> 40	29	9

*Scaled for a 20 kt burst at 1850 ft (0.35 mi) above a sea-level surface.

**The values shown in the body of the table will require adjustment later to be consistent with a refinement of yield estimations for the Hiroshima explosions.

severe injuries and rapid fatalities for some conditions of exposure.

2. The velocity of glass fragments of greater than 400 feet per second, of course, speaks for itself.

3. Displacement velocities of greater than 40 feet per second also are high enough to produce serious injury and early fatality for many geometries of exposure.

4. The thermal flux of 140 cal/cm^2 predicted is very challenging, and the fire hazard would be high. The Post Office building was, in fact, gutted by fire. Too, hot-gas burns are known to have occurred to some individuals. Surprisingly, the acute burn problem could not have been extremely prominent or else 200 individuals would not have survived 20 days.

5. A similar remark may be made concerning the inside radiation dose for there must have been shielding to decrease the computed "free-field" dose of 59,000 by factors of at least 1 in 10 to 1 in 100.

The Japanese data have been presented among other things to emphasize the encouraging fact that survival rates, even inside areas of heavy physical destruction, are higher than one might think from viewing the "free-field" parameters applying to the immediate hazardous effects — this information is worth having.

It is well known that the survivors at Hiroshima mostly walked out of the city after the bombing, and even though a fire storm occurred, about 187,000 of the 255,000 individuals at risk survived, of which about 110,000 escaped uninjured[40, 41]. With considerable emphasis, however, I wish to point out that residual radiation placed no constraints upon the postshot movements of the population and rescue operations; had this been a factor, survival no doubt would have been much depressed.

It is important to recognize even for high-yield surface bursts with unprepared cities as targets that, though casualties would be high, many thousands would escape the immediate effects. These people, both uninjured and injured, must have a place to go and their injuries must be

cared for. Also, since residual radiation occurs very close-in, in time periods like seconds and many tens of minutes for ranges inside those that are hazardous from thermal and blast effects, radiation protective measures for survivors must be provided. Planning must recognize that the structures designed for protection against residual radiation must also be blast and fire resistant if they are to be located, as they should be, within the target area. Can you think of anything more depressing to individuals fortunate enough to survive the blast and thermal effects of a surface burst to their city which had provided planned fallout protection, to make their way as rapidly as possible to the shelter location and find it had been rendered useless by blast and fire?

Let me leave you with this thought while I now turn to the final area of this presentation to make a few remarks about scaling and the comparative variations in major effects as they vary with yield and range.

VI. Scaling "Free-Field" Effects

Figure 45 offers a basis for illustrating the first important points, for it shows the yield-range relationship for arbitrarily chosen separate effects; e. g., 30 and 100 rems, 1 and 5 psi, and 1st and 2nd degree burns. The data were all scaled using information from The Effects of Nuclear Weapons[6].

Note that, in general, the range of each effect increases with yield, but that the relative increase in range is not the same for each effect. Initial radiation increases the least, thermal radiation the most, and the blast pressures in between.

It is important to recognize that any and all of the Japanese data apply only to a small area of the yield-range-effect spectrum – in and about 20 kilotons – and that extrapolation to other yields involves a variety of different comparative relationships.

One such relationship is shown in Figure 46 in which initial "free-field" blast overpressures are shown as a function of the dose of initial ionizing radiation for sea-level surface bursts of 1 and 100 kiloton and

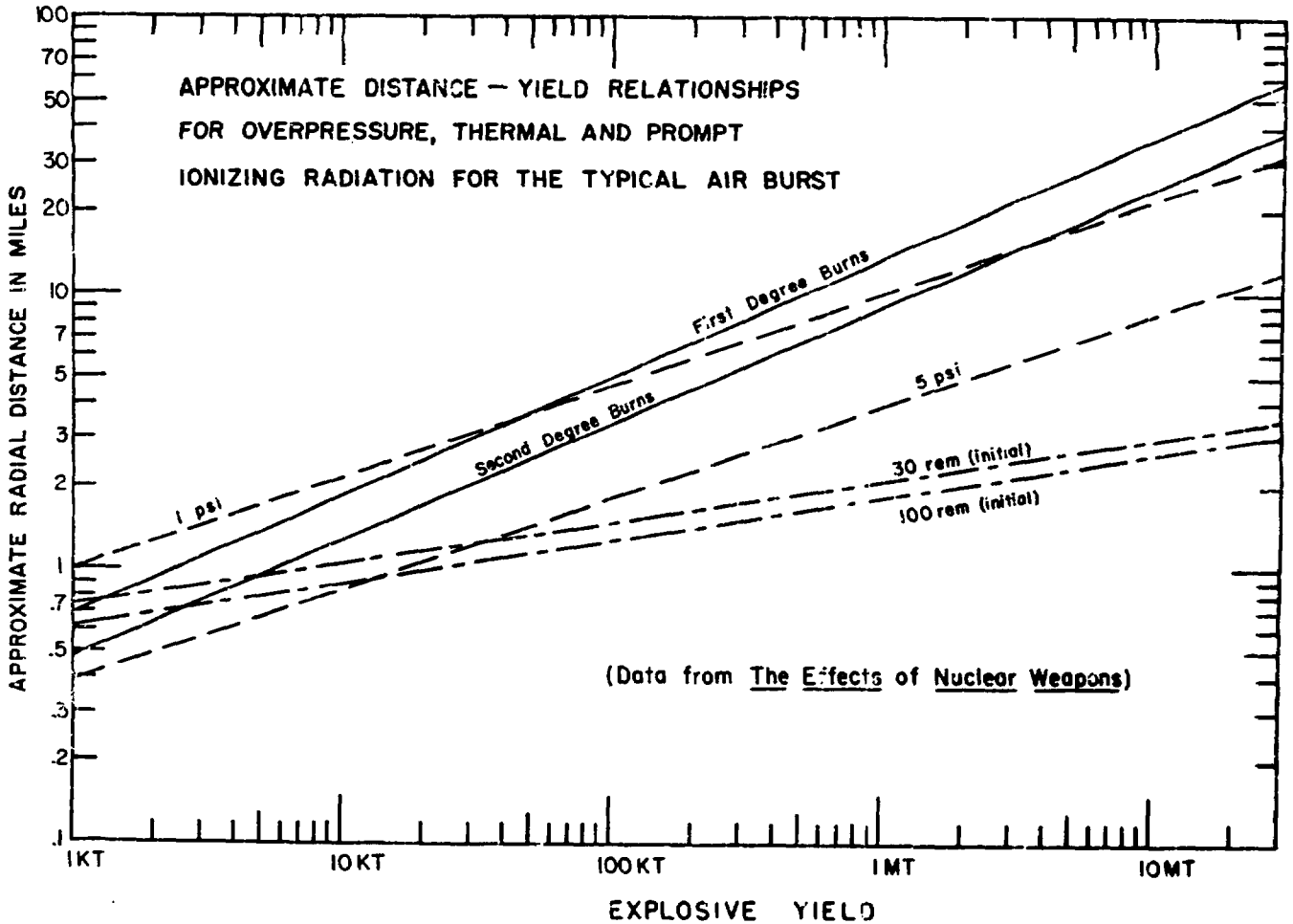

APPROXIMATE DISTANCE — YIELD RELATIONSHIPS
FOR OVERPRESSURE, THERMAL AND PROMPT
IONIZING RADIATION FOR THE TYPICAL AIR BURST
APPROXIMATE RADIAL DISTANCE IN MILES
100
70
50
30
20
10
7
5
3
2
1
.7
.5
.3
.2
.1
First Degree Burns
Second Degree Burns
1 psi
5 psi
30 rem (initial)
100 rem (initial)
(Data from The Effects of Nuclear Weapons)
1KT
10KT
100KT
1MT
10MT
EXPLOSIVE YIELD

Fig. 45

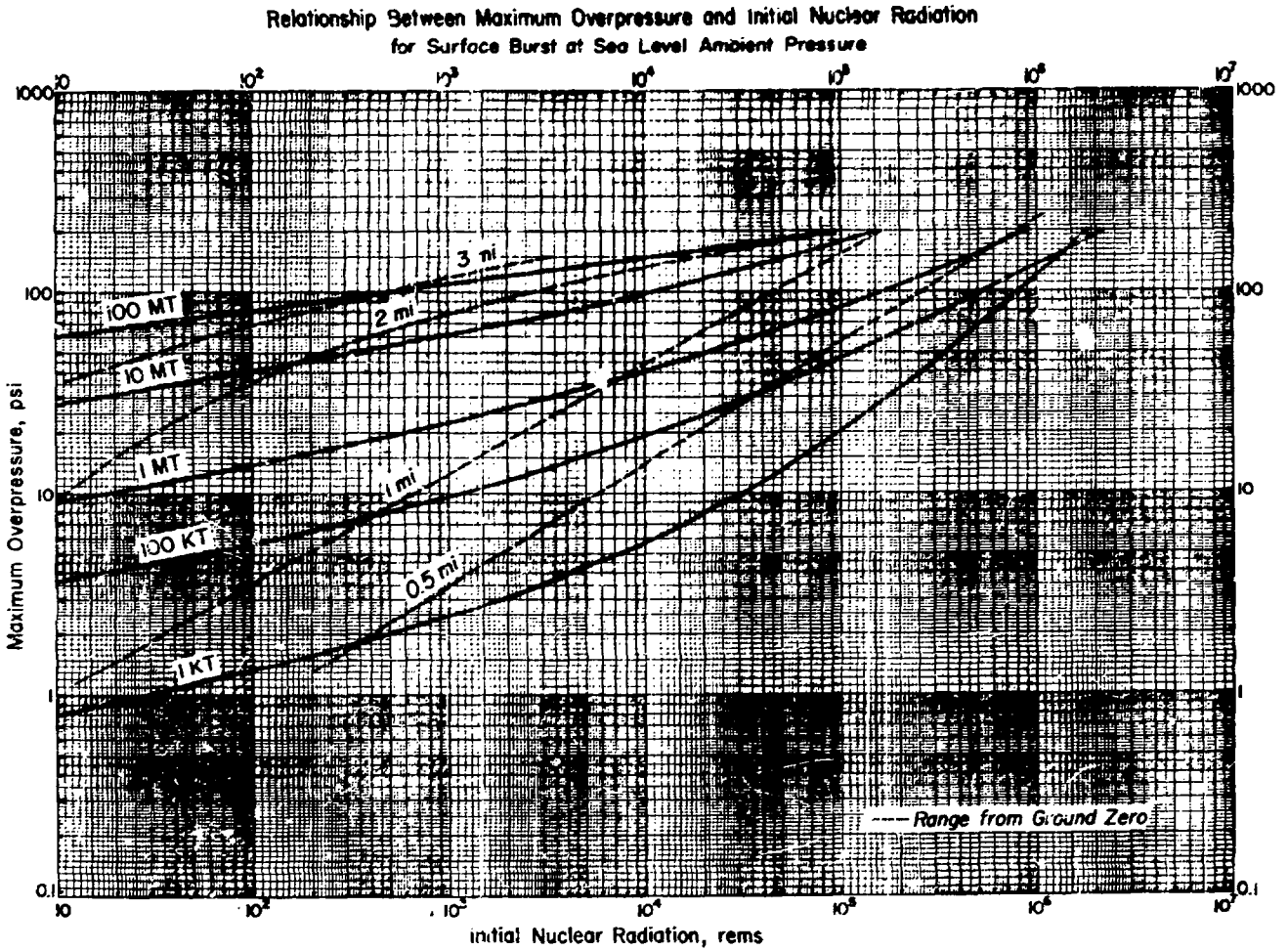

Relationship Between Maximum Overpressure and Initial Nuclear Radiation
for Surface Burst at Sea Level Ambient Pressure
Maximum Overpressure, psi
Initial Nuclear Radiation, rems
100 MT
10 MT
1 MT
100 KT
1 KT
3 mi
2 mi
1 mi
0.5 mi
---- Range from Ground Zero

Fig. 46

1, 10, and 100 megatons. If one considers a dose of 100 rems as acceptable in an emergency, plans for survival at the 100 rem range must consider overpressures of about 14 and 40 psi for the 1- and 10-megaton bursts, respectively.

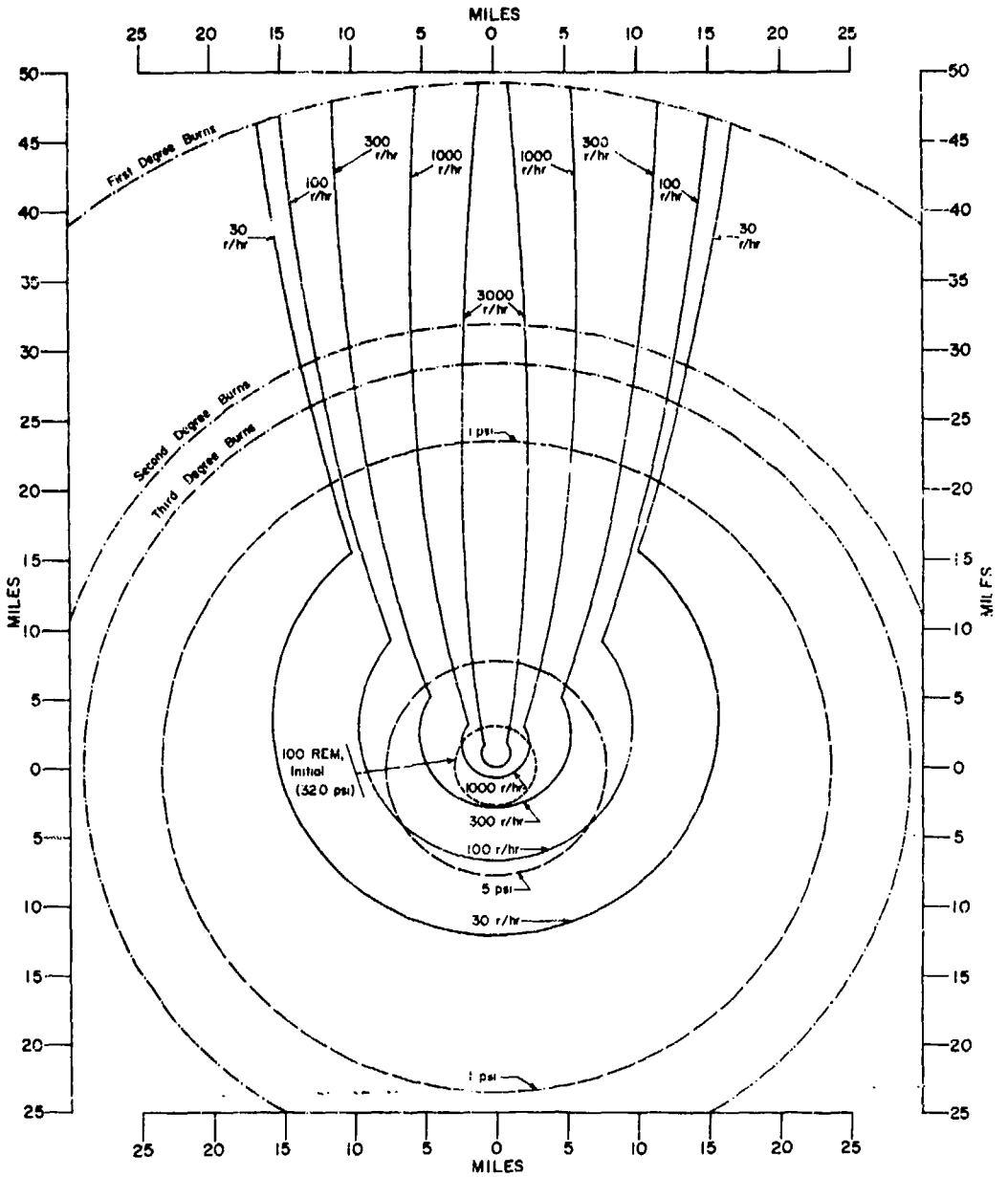
If survival plans included shielding by a factor of 100 from initial ionizing radiation, then reading from the illustration, above the 10^4 figure on the lower scale, one can see that protection from overpressures of near 40 and 100 psi, respectively, for the 1- and 10-megaton bursts would be required. Not to recognize this is about the same as "writing off" all the individuals inside a radius from near 1.0 to 2.1 miles of ground zero. Frankly, the latter to me in high-density areas of population is not at all justifiable, since protection in the Nevada Tests up to 200-250 psi has been proven quite feasible.

Figure 47, scaled for a 20-megaton surface burst at sea level, is presented to help put the over-all problem of survival planning in better perspective[4, 6].

Note that the isodose-rate contours for residual radiation down to 30 roentgens per hour at 1 hour applying to a 15 mile per hour wind only cover about 30-40 degrees of the arc enclosed by the 1st degree burn line of near 50 miles scaled for maximal visibility. This area of risk is far from the 360° arc covered by blast and thermal effects.

However, because in a matter of minutes, there will be significant radiation in and about ground zero including the upwind direction, residual ionizing radiation should be considered one of the early effects of surface bursts. This fact along with the inability to know the direction of the wind ahead of time makes protection against residual radiation a necessity, not only at great range from the burst, but also in the entire area immediate to the target ground zero.

Through systematic planning for protection against the most far-reaching hazard — thermal fluxes — and then blast overpressures and winds, survival in to the 100 rem circle of about 2.4-mile radius where



COMPARATIVE EFFECTS FOR A 20 MT SURFACE BURST

Residual radiation data - one hour reference dose rates - computed for a fission yield of 10 MT and an effective wind of 15 mph

Fig. 47

near 50 psi can be anticipated, seems a quite straight forward problem. If protection against residual radiation is added and sufficient shielding arranged for high fluxes of initial radiation, then survival well inside the fireball radius of about 2.6 miles frankly seems feasible to me. Indeed, if protection up to 200 psi that has been proven feasible in Nevada is taken as a criteria, then one, blast-wise, would contemplate survival into about 1.3 miles even for a surface burst of 20-megaton yield. This would only require initial ionizing radiation shielding by a factor of about 10^3 and about the same degree of thermal protection.

It is encouraging that an optimist like myself can say these things. It is heartening that missile bases are being hardened to withstand high fluxes of all the effects parameters, and that the sea offers shielding for Polaris submarines and personnel. It is, however, downright depressing that an accelerated and systematic program to bring protection to the mass of the population on a national scale has been so long delayed.

SUMMARY

A brief summary of the material covered in this presentation follows:

1. First, following a few introductory remarks, attention was directed to five problem areas of concern to those who think about the biological effects of nuclear weapons; namely, "free-field" and "geometric" scaling, secondary events, etiologic mechanisms, and hazards assessment.

2. Second, the scope of blast biology was defined to include primary or pressure effects, secondary effects due to damage from penetrating and nonpenetrating missiles, tertiary effects as those occurring as a consequence of displacement, and miscellaneous effects due to dust and non-line-of-sight thermal hazards.

3. Third, certain physical and biophysical factors were considered to abet better understanding the environmental variations which follow explosive phenomena and the biophysical events associated with biological damage. These concerned the aerodynamics of displacement of objects

including man and consideration of simple fluid models.

4. Selected experimental data were summarized in the area of biological response mostly related to primary and tertiary blast effects. The overpressure-time relationship for "sharp"-rising pressures as it influences lethality for large and small animals, including estimates for man, was reviewed and the significance of stepwise and slowly-rising increases of overpressure was noted. Also, pathophysiologic data responsible for lethality were presented and the results of an interspecies impact study were set forth.

5. Fifth, biologic criteria considered "safe" for emergency conditions – see Table 6 – were noted as were the environmental variations likely to be associated with significant casualties and lethality.

6. Sixth, selected survival data from Hiroshima were compared to show the marked variation in 50 per cent survival ranges as conditions of exposure varied.

7. Seventh, a few remarks were made about "free-field" scaling and the comparative variations in the major effects which can be anticipated as functions of yield and range. Also, the data were used to show that measures which are effective against the most far reaching effects result in a relative change in the environmental challenge at locations closer to ground zero.

8. Eighth, the need for planning sound protective measures against all hazardous weapons effects was emphasized as one of the most attractive alternatives for minimizing casualties and maximizing survival.

9. Ninth and finally, four additional points were made; namely, (a) let those who grasp the implications of biological blast effects add them to the hazards from thermal and ionizing radiation; they cannot escape being concerned because insufficient local, regional, and national attention is being paid to the immediate biological effects of nuclear weapons; (b) arranging survival of many millions of people in case of a nuclear war is not as technically difficult as most individuals believe it to be; (c) it is

TABLE 6

Biological Criteria Considered "Safe" for Emergency Conditions

Major Effect	Critical Organ Or Event	Value for Allowable Acute Environmental Variation
Blast*		
Overpressure	Lungs	15 psi maximal incident overpressure with a classical wave form
		6 psi incident reflecting "instantaneously" to 15 psi maximal
	Ears	5 psi incident and maximal overpressure
		2.5 incident reflecting to 5 psi maximal
Missiles	Penetration into a serous cavity	100 ft/sec for a 10 gm glass missile
	non-penetrative skull fracture	10 ft/sec for a 10 lb blunt object
Displacement	Skull fracture from impact	10 ft/sec for 160 lb man
Ionizing Radiation	Whole body	Up to 150-200 rem.
Thermal Radiation	Uncovered white skin	1st degree burn - 2.0-5.0 cal/cm^2 depending on yield

*Applies to "sharp"-rising overpressures enduring for 100 msec or longer; i.e., mostly for yields above 1 KT.

high time the aura of gloom which surrounds nuclear arms be destroyed. Let us substitute the impetus planned and implemented measures to enhance survival can give nuclear deterrence and let the latter be welcomed as a significant addition to national security; and (d) fourth, with regard to the social and political implications of the last statements, let me say I am convinced the society which first makes these adaptations at the thinking and working level will without question eventually control the lives of men on this planet.

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